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Development and Evaluation of Rehabilitation Technologies for Early-Stage Spinal Cord Injury

Colm Tadhg Donal Craven, BE MEngSc

A thesis submitted for the degree of
Doctor of Philosophy (PhD)

School of Engineering
College of Science and Engineering
University of Glasgow

December 2014

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Author's Declaration

I declare that, except where explicit reference is made to the contribution of others, that this thesis is the result of my own work and has not been submitted for any other degree at the University of Glasgow or any other institution.

All of the analysis and data collection techniques presented in this thesis were created by the author.

C.T.D. Craven
December 11, 2014

Abstract

Exercise is fundamental to a return to normal living after a Spinal Cord Injury (SCI) but is complicated by a rapid decline in function and fitness immediately following the injury. In addition to muscle paralysis, Orthostatic Hypotension (OH), a decline in cardiopulmonary and vascular fitness, extensive muscle atrophy and bone demineralization each contribute to an inability to carry out effective forms of exercise. Suitable rehabilitation technologies are central to overcoming these complications and to facilitate recovery. The aim of the work presented in this thesis was to identify technologies which may be used for exercise during SCI rehabilitation, with a focus on early-stage SCI patients at the beginning of the rehabilitation process. Two such technologies were identified and investigated: Robotic Assisted Tilt Table Therapy (RATTT) and Whole Body Vibration (WBV). The suitability of these technologies was evaluated by measuring the acute physiological responses of SCI patients during intensive use of these systems.

Robotic Assisted Tilt Table Therapy is primarily used to treat OH in stroke and SCI patients. However, the potential therapeutic effect of RATTT as an exercise modality has so far not been investigated. An investigation into the physiological responses of early-stage SCI patients during intensive RATTT stepping was therefore carried out. The intention was to determine whether RATTT may potentially be used for the combined purpose of increasing orthostatic tolerance, improving cardiopulmonary fitness and improving vascular health. RATTT is particularly suited to early-stage rehabilitation because orthostatic tolerance is not a prerequisite for its use. Three motor-complete and three motor-incomplete early-stage SCI patients were recruited from the Queen Elizabeth National Spinal Injuries Unit (QENSIU) in Glasgow to participate in this cross-sectional study. The cardiopulmonary and vascular responses to different RATTT stepping conditions, including the combination of RATTT stepping with Functional Electrical Stimulation (FES), were investigated. Increases in oxygen uptake, respiratory exchange ratio, minute ventilation and heart rate were found for both motor-incomplete and motor-complete SCI participants. The responses were largest for the motor-incomplete volunteers with Metabolic Equivalent (MET) values between 1.5 - 3.1. These results confirmed that RATTT can be used as an exercise modality during early-stage SCI rehabilitation.

Whole Body Vibration was initially developed and commercialised for application during exercise in a general population with no neurological deficit, and while the physiological mechanisms which underly the response to WBV are still relatively poorly understood, current research suggests that WBV may lead to increases in muscle strength, power, bone mineral density and flexibility. It is hypothesised that the neuromuscular response to WBV is achieved through reflex activity, though the specific neural pathway is broadly debated. Nonetheless, an increase in neuromuscular activity during WBV has been confirmed, suggesting that it may potentially be used to increase muscle mass, strength and power, and therefore counteract muscle atrophy and bone demineralization in SCI. However, little is known about the neuromuscular response of SCI patients to WBV, and it is not clear how to best administer WBV to this patient group or which vibration parameters should be applied. A WBV platform was therefore integrated with a partial Body Weight Support (pBWS) system in order to investigate the application of WBV during SCI rehabilitation.

The feasibility of this approach was determined in the first instance in experiments with participants from a general population with no neurological deficit, followed by an evaluation with a SCI population. The aim was to determine if the stimulus from WBV applied in conjunction with pBWS was sufficient to elicit an increased neuromuscular response, and if so, to characterise the magnitudes and trends of the responses.

Ten participants with no neurological deficit were recruited to investigate the feasibility of WBV-pBWS and to establish a normative data set with which to compare the results from 14 SCI participants recruited from QENSIU. The main factors under investigation were vibration frequency, vibration amplitude, level of pBWS, muscle group and classification of SCI. It was shown that WBV did elicit an increase in neuromuscular activity and that the magnitude of the response could be moderated by vibration frequency, vibration amplitude and level of pBWS. Average changes relative to baseline measurements were up to 71% for the neurologically intact participants, and between 44% to 66% for the SCI participants depending on classification of injury. Neuromuscular activity was characteristic for each muscle group and the characteristic was principally moderated by the proximity of the muscle to the WBV platform and peak platform acceleration. Despite the relatively large change in neuromuscular activity when compared to baseline, the absolute changes in activity were relatively small and likely to be of insufficient magnitude to result in muscle hypertrophy. Results from this study indicated that WBV was of sufficient intensity to elicit a response from the α -motoneuron but of insufficient intensity to increase muscle strength. Based on this, the potential use of WBV as a non-pharmacological treatment of spasm was identified by stimulating part of the neural pathway upon which spasm acts and therefore provide a mechanism to moderate the threshold for spasm, without the risk of increasing muscle strength and therefore potential for injury during a spastic episode.

In summary, this thesis presents RATTT and WBV-pBWS as two modalities suitable for use in early-stage SCI rehabilitation. While RATTT can elicit substantial cardiopulmonary responses in this patient group, the evaluation of WBV showed limited effects on muscle activation, but suggested potential application in the treatment of spasticity.

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Go raibh míle maith agaibh go léir!

(Frank, this thesis is 250 pages long - I hope this meets with your approval.)

Thesis Outline

- **Chapter 1:** This chapter introduces the physiological background of the spinal cord and Spinal Cord Injury (SCI). The normal function of the central nervous system is disrupted after a SCI and the prevalence, etiology, and consequences of a SCI are therefore described. Some of the chronic complications that arise secondary to a SCI are outlined, emphasising the importance of developing exercise technologies and protocols, which facilitate SCI rehabilitation. Two such technologies which were researched in detail for this thesis are Robotic Assisted Tilt Table Therapy (RATTT) and Whole Body Vibration (WBV) with partial Body Weight Support (pBWS). The underlying principals of the measurement systems used to assess the physiological responses of neurologically-intact and spinal cord injury volunteers to these rehabilitation technologies, which included metabolic rate (pulmonary gas exchange) and neuromuscular activity (EMG activity), are described.
- **Chapter 2:** RATTT was investigated to determine its potential application for exercise during SCI rehabilitation. The study was carried out with a cross-sectional sample taken from an early-stage (<1 year) SCI population with no lower motor neuron damage. The potential therapeutic effect of RATTT was determined by assessing the cardiopulmonary and vascular responses during different stepping conditions for motor-incomplete SCI and motor-complete SCI participants. The results were compared to exercise loads in the general population by calculating the metabolic equivalent (MET).
- **Chapter 3:** This chapter presents a review of the literature on WBV. The review initially focuses on the acute responses of those from the general population with no neurological deficit and those with a SCI to WBV. Following this, the main theories describing how WBV elicits an increased neuromuscular response are described. Studies investing the chronic changes in muscle after a period of WBV training are presented, with a focus on changes in muscle strength and muscle power.
- **Chapter 4:** The experimental methods and protocols used for the WBV studies outlined in Chapters 5 & 6 are presented, with focus on the experimental setup of WBV-pBWS system and the resultant data analysis techniques.
- **Chapter 5:** This chapter presents the first stage of the WBV-pBWS investigation which was carried out with a sample from the general population with no neurological deficit. A general population was used in the first instance to establish a normative baseline with which to compare the response of those with a SCI, and to establish a suitable protocol for application in SCI. The acute neuromuscular response of the lower limb to different WBV conditions and levels of pBWS were investigated using Electromyography (EMG) measurements.
- **Chapter 6:** This chapter presents the second stage of the WBV-pBWS investigation which was carried out with a cross-sectional sample taken from a SCI population. The experimental protocol was informed by the findings from the first stage, with variations applied to take account of the unique challenges presented by those with a SCI. The acute neuromuscular response of the lower limb muscles to different WBV conditions were again investigated using EMG. A case study is included in this chapter

which investigates the measured electromyographic response from a SCI participant with flaccid paralysis in order to validate some of the offline filtering techniques used throughout this thesis.

- **Chapter 7:** This chapter summarises the conclusions of the thesis and outlines future work. Observations from both WBV-pBWS studies indicated an increased neuromuscular response to WBV. The relatively small changes found in SCI may preclude the use of the technology for increasing the mass or strength of paralysed muscle, however other equally important uses of the technology for SCI rehabilitation are discussed which include the treatment of spasm. Suggestions are made for future improvements to the system design.

Contributions

1. This thesis presents the first research study which investigated the potential therapeutic benefit of Robotic Assisted Tilt Table Therapy (RATTT) for cardiopulmonary exercise in a Spinal Cord Injury (SCI) population. A novel feedback system was developed and used to encourage greater voluntary participation in the stepping exercise.
2. The RATTT study was the first to directly compare the vascular and cardiopulmonary responses of both motor-complete and motor-incomplete SCI participants during non Functional Electrical Stimulation (FES) supported, and FES-supported, RATTT stepping.
3. A new system was developed which allowed the application of Whole Body Vibration (WBV) to a SCI population by combining it with a partial Body Weight Support (pBWS) system. WBV was applied in a standing position rather than on a tilt-table, standing frame or from a chair as was done previously. This novel system allowed the examination of the neuromuscular response of both neurologically-intact and SCI volunteers using similar experimental protocols.
4. This research was the first to investigate and describe the acute, steady-state neuromuscular response of a general population with no neurological deficit to a broad range of vibration conditions at reduced load. Changes in loading during previous applications of WBV were achieved only by increasing the supported weight carried by the participants.
5. This research assessed the feasibility of applying WBV to a SCI population and quantified the acute, steady-state neuromuscular response. The investigation was the first to quantitatively determine and compare the acute neuromuscular responses of early-stage (<1 year) motor-complete and motor-incomplete SCI populations to a broad range of vibration conditions in a standing position.
6. Vibration artefact was detected in the Electromyogram (EMG) recorded during WBV. An appropriate filtering technique was established through statistical analysis and validated by testing on EMG recorded from a SCI participant with flaccid paralysis.
7. Precision acceleration measurements were used to accurately determine the specific WBV platform position throughout each test. A novel analysis approach was used which combined acceleration and EMG measurements to qualitatively describe the neuromuscular responses of the entire sampled population during a single cycle of the WBV platform. These findings support the hypothesis that the neuromuscular response to WBV is moderated by peak acceleration.

Publications

- C.T.D. Craven, H. Gollee, S. Coupaud, M.A. Purcell, and D.B. Allan, ‘Investigation of the acute neuromuscular response to whole body vibration with partial body weight support’, In preparation.
- C.T.D. Craven, H. Gollee, S. Coupaud, M.A. Purcell, and D.B. Allan, ‘Investigation of robotic assisted tilt-table therapy for early stage spinal cord injury rehabilitation’, *Journal of Rehabilitation Research and Development*, 50(3), pp. 367-378, 2013.
- C.T.D. Craven, H. Gollee, S. Coupaud and D.B. Allan, ‘The application of FES during whole body vibration with body weight support: a pilot study’, in *Proceedings of the 3rd Annual International Functional Electrical Stimulation Society Conference (UKROI Chapter)*, Dublin, Ireland, March 2011.
- C.T.D. Craven, H. Gollee, M.A. Purcell, and D.B. Allan, ‘Cardiopulmonary responses to active and electrically stimulated stepping, with robotic assistance in early-stage spinal cord injury, in *Proceedings of the 15th Annual International Functional Electrical Stimulation Society Conference*, Vienna, Austria, September 2010.

List of Abbreviations

ACSM	American College of Sports Medicine
ADP	Adenosine Diphosphate
AIS	ASIA Impairment Scale
ANOVA	Analysis of Variance
ASIA	American Spinal Injury Association
ATP	Adenosine Triphosphate
BF	Biceps Femoris
BMD	Bone Mineral Density
BTR	Burst-to-Tonic Ratio
CMAP	Compound Motor Action Potential
CNS	Central Nervous System
CSCI	Motor Complete Spinal Cord Injury
DVT	Deep Vein Thrombosis
EMG	Electromyography
ES	Effect Size
FES	Functional Electrical Stimulation
HR	Heart Rate
ISCI	Motor Incomplete Spinal Cord Injury
ISEK	International Society of Electrophysiology and Kinesiology
L-GA	Lateral Gastrocnemius
LMN	Lower Motor Neuron
MAP	Mean Arterial Pressure
MET	Metabolic Equivalent
M-GA	Medial Gastrocnemius
MinVen	Minute Ventilation
MS	Multiple Sclerosis
MUAP	Motor Unit Action Potential
MVC	Maximum Voluntary Contraction
NT	Not Testable
OH	Orthostatic Hypotension
pBWS	partial Body Weight Support
PSD	Power Spectral Density
RATTT	Robotics-Assisted Tilt-Table Therapy
RER	Respiratory Exchange Ratio
RF	Rectus Femoris
RMS	Root Mean Square
SA	Side-Alternating
SCATS	Spinal Cord Assessment Tool for Spastic Reflexes
SCI	Spinal Cord Injury
TA	Tibialis Anterior
TBI	Traumatic Brain Injury
TSD	Time Series Data
TSI	Time Since Injury
TVR	Tonic Vibration Reflex
UMN	Upper Motor Neuron
VL	Vastus Lateralis
VO	Oxygen Uptake
VV	Vertically-Vibrating
WBV	Whole Body Vibration
WHO	World Health Organisation

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Chapter 1

Introduction

This thesis presents two studies that have investigated exercise technologies and their potential application in Spinal Cord Injury (SCI) rehabilitation. Methods for the measurement and interpretation of these physiological changes are outlined. The effect of damage to the spinal cord is described, outlining how SCI is classified and describing some of the primary and secondary complications that arise. The reader is referred to the appendix for a description of the basic anatomy, structure, and function of the spinal cord and nervous system, along with a description of how communication within the nervous system occurs, and how this allows movement to occur via motor action from muscle fibers and their sensory response.

1.1 Physiological Measurements of Muscle

Exercise is carried out by increasing volitional movement at an increased rate or load, therefore increasing the stress placed on the cardiopulmonary and respiratory systems. To predict the efficacy of a particular form of exercise it is necessary to measure and record the magnitude of muscle cell activity during exercise. Two suitable techniques that may be employed are: (i) EMG, which directly measures neuromuscular activity, and (ii) pulmonary gas exchange analysis which can be used to infer cell metabolism. Each of these techniques are used in the studies described in this thesis. The studies investigate the potential benefit of new rehabilitation techniques for exercise in SCI patient populations.

1.1.1 Electromyography

Electromyography is the recording of the Motor Unit Action Potential (MUAP) generated by a muscle by placing electrodes in the immediate vicinity of the muscle fiber. MUAP are small, and are normally in the μV scale. An electromyogram is produced by EMG and is described as a graphical representation of neuromuscular activity. To record an EMG using modern recording equipment, it is necessary to amplify the MUAP in order to detect it. The most common type of amplifier is the differential amplifier. Differential amplification requires the placement of two detection electrodes in parallel with the muscle fiber. A reference electrode is placed at an electrically inactive region of the body, away from the muscle fiber (Figure 1.1). Common background signals are detected by the reference electrode and subtracted from detection electrodes. The amplifier also subtracts the signals recorded from the two detection electrodes from each other. This allows for the detection of the localised potential

difference between the two electrodes.

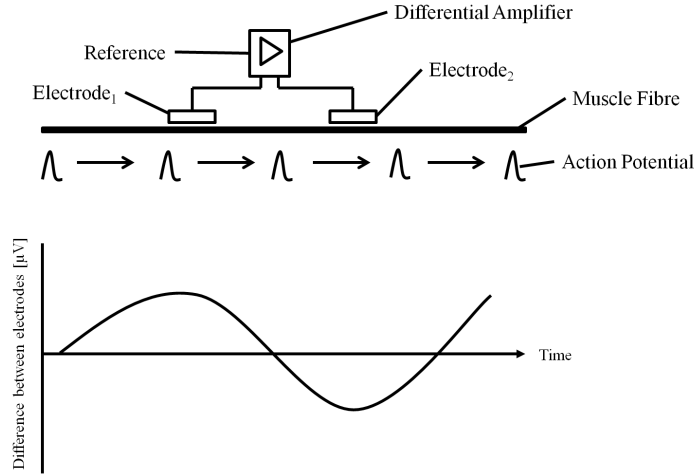


Figure 1.1: Schematic of a single motor unit action potential detected by electromyography. An action potential propagating along the sarcolemma of the muscle fibre is first detected by Electrode₁. The potential difference reaches a maximum as it passes the first electrode, zero when it is equidistant to both electrodes and a minimum as it passes the second electrode. For this reason the action potential creates a bipolar output when it is detected in this way.

A single motor unit action potential can be detected using fine-wire EMG. A needled electrode inserted into the belly of the muscle can be used to detect a MUAP as it propagates along the sarcolemma of the muscle fibre. Fine-wire electromyography is particularly suited to studying the physiology and pathology of individual motor units. Alternatively, the activity of a several motor units (Compound Motor Action Potential (CMAP)), can be recorded using surface EMG electrodes. During surface EMG, the detection electrodes are affixed to the skin over the belly of the muscle rather than inserted into the muscle. Surface EMG is better suited for investigating more general aspects of neuromuscular activity, such as activation timing during dynamic contractions, temporal activity, or fatigue. The output will be a summation of the individual MUAP's due to the spatial distribution of the muscle fibres with respect to the detection electrodes as described in Figure 1.2 below.

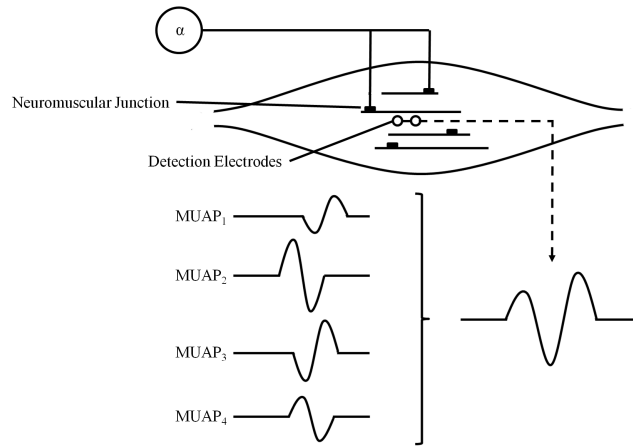


Figure 1.2: Schematic outlining the superposition of several action potentials propagating along different muscle fibres within a single motor unit. The action potential detected by the detection electrodes placed over the surface of the muscle record a potential difference that represents the summation of the individual action potentials for each muscle fibre.

1.1.2 Pulmonary Gas Exchange Analysis

Adenosine Triphosphate (ATP) production can be anaerobic or aerobic. At the onset of exercise, phosphocreatine may be used to produce ATP in the absence of oxygen. However, there is a limited store of phosphocreatine in the muscle and alternative production methods are required which include glycolysis and oxidative phosphorylation. ATP and pyruvic acid are produced during glycolysis. In the absence of oxygen, pyruvic acid is converted into lactic acid. Oxygen is also required to convert lactic acid back into pyruvic acid during rest and recovery. During oxidative phosphorylation, oxygen and pyruvic acid are ultimately converted into energy which is used to produce ATP, carbon dioxide, and water. The supply of oxygen and the disposal of carbon dioxide is therefore central to sustained muscular activity, and are linked to respiration as described in Figure 1.3.

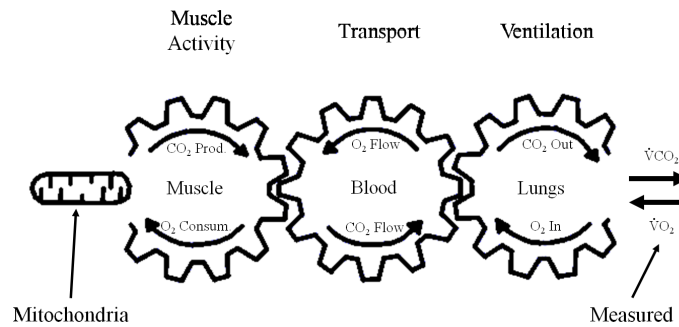


Figure 1.3: Schematic describing the coupling of respiration at the pulmonary and cellular level (adapted from Wasserman *et al.* [1]). Oxygen consumed, and carbon dioxide produced at the cellular level are transported by the blood via gas exchange at the lungs to the mouth. Therefore, recording minute ventilation of oxygen ($\dot{V}O_2$) and carbon dioxide ($\dot{V}CO_2$) at the mouth may be used to infer the muscle metabolism at the cellular level.

Inspired oxygen ($\dot{V}O_2$) and expired carbon dioxide ($\dot{V}CO_2$) are transported via the

circulatory system to and from the mitochondria. These parameters can therefore be measured at the mouth to infer that which is occurring at the cellular level within the muscle.

1.2 Spinal Cord Injury

A Spinal Cord Injury (SCI) causes damage to the spinal cord resulting in extensive disruption of the central nervous system leading to significant, global health complications. The damage to white and grey matter severely disrupts the passing of motor and sensation signals to and from the brain. The primary consequence of SCI is therefore paralysis of the muscles below the level of lesion. In some cases the degree of paralysis is minor and significant functionality may be regained with appropriate health care, management, and physical rehabilitation. In other cases, the degree of paralysis is severe, and the patient will require a lifetime of extensive care and management.

SCI is most commonly the result of a trauma caused by a road traffic incident, fall, or penetrating injury [2]. Depending on the level and severity of the SCI mobility, breathing, sexual function, bowel function and bladder function can be compromised. Autonomic disruption may affect the regulation of heart rate, blood pressure, breathing and body temperature. High cervical injuries result in compromised motor function of the upper limb, torso and lower limb, and ventilator dependence due to paralysis of the breathing muscles is common. Low cervical to mid thoracic injuries result in some preserved upper limb and torso strength but disrupted autonomic function. Low thoracic and high lumbar injuries result in slightly compromised upper body strength but autonomic function is normal. Low lumbar and sacral injuries result in normal upper body strength but in some cases areflexia of the lower limb, bowel and bladder occurs.

These severe complications are likely the reason that tetraplegia was initially described as “*an ailment not to be treated*”¹ when first diagnosed by an Egyptian physician around 2500BC. More recently during World War 1, 10% of patients survived the first year of cord injury, reducing to 1% after 20 years of injury. However, today with appropriate health care and management life expectancy is almost normal.

It is now common for the immediate period after cord injury to be sedentary. Most patients will be surgically stabilised and immobilised after a traumatic SCI. Immobilisation is necessary to allow skeletal deformities to repair and therefore minimises the risk of further neurological damage from an unstable fracture. There is a series of chronic complications which arise subsequently and are secondary to the cord injury. Such complications include muscle atrophy, bone demineralisation, spasticity, pressure sores, orthostatic hypotension, and autonomic dysreflexia. Paralysis and immobilisation significantly contribute to the onset of these secondary health complications. The inability to partake in effective physical exercise delays rehabilitation and minimises the chance of regaining functionality. The risk of pneumonia, septicemia, pulmonary emboli, obesity and death from cardiovascular attack therefore increases.

¹A. Swain, *ABC of spinal cord injury*, BMJ Publishing Group, 4th ed., 2002.

Re-mobilisation and exercise is primarily delayed due to immobilisation which facilitates the onset of orthostatic intolerance. Compromised vasomotor control and orthostatic intolerance result in an inability to participate in intensive exercise. Specialised rehabilitation is therefore required to achieve orthostatic tolerance, therefore delaying participation in exercise activities further. Once orthostatic tolerance is achieved, disuse induced muscle atrophy leads to an inability to participate in prolonged periods of exercise due to muscle fatigue. The inability to exercise contributes to an additional decline in cardiopulmonary fitness, an increase in the prevalence of obesity, and an increase in the risk of cardiovascular attack.

Muscle atrophy is linked to the decline in cardiopulmonary health. The inability to utilise to the large, oxidative muscles of the body reduces the demand normally placed on the cardiopulmonary and vascular systems, therefore resulting in the decline in their function. To break this cycle there is now a focus on the utilisation of technology to facilitate rehabilitation and exercise in the early-stages of SCI, before the onset of secondary health complications.

1.2.1 Overview, Prevalence & Etiology

99 patients were diagnosed with a SCI in Scotland in 2011/12. This was reported to be consistent with the previous 5 years, where the year on year average of new injuries is 104 per year [3], or approximately one injury per 50,000 population. There is a reasonably even distribution in the type of injury, where it was reported in the latest Queen Elizabeth National Spinal Injuries Unit for Scotland Annual Report that, of the new SCI cases reported in 2011/12, 29 were classed as complete tetraplegia, 31 were complete paraplegia, and 39 were incomplete tetraplegia or paraplegia. Across all age groups, more males than females suffer a neurological cord injury. In general there is a reasonably even spread in the number of injuries across all age groups, with a slight peak in the 20 - 29yrs, and 70 - 79yrs age groups [3]. There has traditionally been a larger number of injuries in the younger age groups [4]. However, the increase in the number of injuries now being found in those in the older age groups has been attributed to an increase in the number degenerative spinal fractures, whose incidence increase with age [3].

In Scotland, the highest cause of all injuries is falls (55%), followed by road traffic incidents (23%), with the remainder being made up by sporting injuries, assault, penetrative injuries, self harm, and disease [3]. In a recent report by the National Spinal Cord Injury Statistical Centre, which holds a database of patients with SCI in the USA, the main causes of injury are a reverse of those reported for Scotland. 40% of injuries were a result of road traffic incidents, and 30% a result of falls in 2005 [5].

Mortality is at its highest within the first year of injury, with life expectancy generally increasing thereafter. With appropriate medical treatment, management, and lifelong care, life expectancy is now approaching normal levels. As a result, the leading cause of death in SCI is now pneumonia and septicemia [5,6], in addition to some of the leading causes of death in the general, non-SCI, population such as cardiovascular disease [7].

The typical length of stay in acute care and rehabilitation post-injury typically depends on severity of cord injury. The average length of stay for those with complete tetraplegia is 170

days (Range: 4 - 434 days), complete paraplegia is 119 days (Range: 16 - 293 days), and incomplete tetraplegia or paraplegia is 31 days (Range: 1 - 218 days) [3]. This is considerably different to the length of stay reported for those in the USA, where the average length of stay is 11 days [5]. The large difference in the reported length of stay is attributed to the different philosophies of treatment and rehabilitation practiced in the different countries.

In Scotland, it is reported that the majority of patients do not require re-admission when discharged. Where re-admission is required, it has largely been due to skin problems manifested in the form of decubitus ulcers. The main health problems that SCI patients report, but which do not require re-hospitalisation include: pain and/or spasm (57%), halo fixation (33%), and urology problems (5%), with the remainder made up by skin problems, sexual dysfunction, and fertility issues.

SCI, while uncommon, considerably affects an individual's health, social life and work status. The complexity and severity of this type of injury requires specialised and lifelong after care. The following sections provide a brief overview of how SCI is classified and the type of complications that arise secondary to a cord injury.

1.2.2 Classification

The American Spinal Injury Association (ASIA) 'standard neurological classification of spinal cord injury' is a commonly used technique to determine the extent of neurological deficit that has occurred as a result of a SCI. Pin prick & soft touch sensation, muscle power, reflexes and cranial nerve function are tested. Sensation at the dermatomes identified in Figure 1.4 are classed as 0 (absent), 1 (partial), 2 (normal) or Not Testable (NT) [8]. The power of key muscle groups, which include the: elbow flexors and extensors, wrist extensors, finger flexors and small finger abductors, hip flexors, knee extensors, ankle dorsiflexors, long toe extensors and ankle plantar flexors tested are also tested. Muscle strength is graded as 0 (total paralysis), 1 (visible contraction), 2 (gravity eliminated full range of motion), 3 (full range of motion against gravity), 4 (full range of motion against some resistance), 5 (normal, full range of motion against full resistance) or NT [8]. These dermatomes and myotomes have been chosen because they are most easily accessed in a supine position. Sacral segments are tested by digitally checking mucocutaneous and deep anal sensation and voluntary anal contraction [9]. Based on these tests, the neurological level of lesion is defined as the most caudal segment of the spinal cord at which sensory and motor function is normal. In some cases, the neurological level of injury can be quite different to the level at which the largest degree of vertebral damage has occurred, in the case of vertebral damage due to trauma.

If there is no motor or sensory function below the neurological level of lesion, the injury is described as being 'complete'. If some sensory or motor function is preserved below the level of lesion, the injury is described as 'incomplete'. In some cases, it is possible for an injury to be motor complete but sensory incomplete (Table 1.1). Depending on the degree of preserved function, or lack thereof, the 5-point ASIA Impairment Scale (AIS) is used to classify the injury [8]. Paralysis that affects all four limbs is described as tetraplegia, and paralysis that affects the lower limbs only is described as paraplegia [10].

Patient Name _____
 Examiner Name _____ Date/Time of Exam _____

ASIA **STANDARD NEUROLOGICAL CLASSIFICATION OF SPINAL CORD INJURY** **ISC**

MOTOR
 KEY MUSCLES (scoring on reverse side)
 R L
 C5 Elbow flexors
 C6 Wrist extensors
 C7 Elbow extensors
 C8 Finger flexors (distal phalanx of middle finger)
 T1 Finger abductors (little finger)
 UPPER LIMB TOTAL (MAXIMUM) (25) (25) (50)
 Comments:
 L2 Hip flexors
 L3 Knee extensors
 L4 Ankle dorsiflexors
 L5 Long toe extensors
 S1 Ankle plantar flexors
 Voluntary anal contraction (Yes/No) _____
 LOWER LIMB TOTAL (MAXIMUM) (25) (25) (50)
 TOTALS (MAXIMUM) (50) (50) (100)

SENSORY
 KEY SENSORY POINTS
 0 = absent
 1 = impaired
 2 = normal
 NT = not testable
 LIGHT TOUCH R L R L
 PIN PRICK R L R L
 Any anal sensation (Yes/No) _____
 PIN PRICK SCORE (max: 112)
 LIGHT TOUCH SCORE (max: 112)
 NEUROLOGICAL LEVEL R L
 SENSORY MOTOR
 COMPLETE OR INCOMPLETE? _____
 ASIA IMPAIRMENT SCALE
 ZONE OF PARTIAL PRESERVATION
 SENSORY MOTOR R L

Figure 1.4: Standard neurological classification of spinal cord injury [8].

Table 1.1: ASIA Impairment Scale. (Adapted from May *et. al.* [8])

Score	Description
A	Complete. No sensory or motor function preserved in the sacral segments S4 - S5.
B	Incomplete. Sensory but no motor function preserved below neurological level of lesion, including the sacral segments S4 - S5.
C	Incomplete. Sensory and motor function preserved below the neurological level of lesion. More than half of the key muscles below the level of have a muscle score < 3.
D	Incomplete. Sensory and motor function preserved below the neurological level of lesion. More than half of the key muscles below the level of lesion have a muscle score ≥ 3 .
E	Normal. Sensory and motor function is normal.

1.2.3 Spinal Syndromes

In addition to ASIA classification, the damage caused by a SCI can generally be described by one of a number of different syndromes, depending on the level and degree of damage done to the ascending sensory and motor tracts. An anterior cord syndrome generally results in preserved proprioception but reduced motor function and sensitivity to pain and temperature (Figure 1.5(a)) [8]. A central cord syndrome generally results in a weakened upper limb compared to the lower limb and sacral sparing (Figure 1.5(b)) [8]. A posterior cord syndrome generally results in good power, and pain and temperature sensation but diminished proprioception (Figure 1.5(c)) [8]. Brown-Sequard syndrome arises when damage occurs to a hemisphere of the spinal cord and generally results in ipsilateral proprioception and motor loss, and diminished contralateral pain and temperature sensitivity (Figure 1.5(d)) [8]. These syndromes are all described as upper motor neuron lesions.

Conus medullaris and cauda equina syndromes result in damage to the lumbar and sacral spinal roots resulting in a loss of bowel, bladder and lower limb reflexes [9]. These syndromes are described as lower motor neuron lesions as they result in areflexia. In some cases, if the damage to the conus medullaris is sufficiently caudal, the damage resembles an upper motor neuron lesion.

The neurological deficit and compromised functional ability of this patient population leads to a number of neurological, skeletal and muscular changes that require careful management and rehabilitation to minimise the number, and delay the onset, of subsequent chronic health complications.

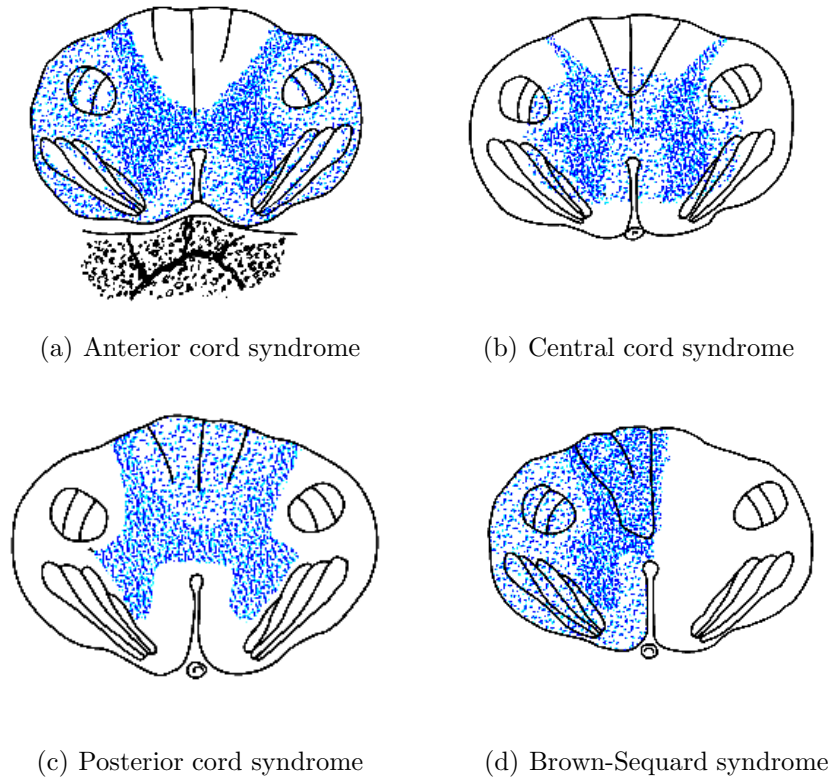


Figure 1.5: Upper motor neuron syndromes (Adapted from Swain *et. al.* [9]).

1.2.4 Chronic Complications

In addition to neurological impairment culminating in paralysis and compromising the normal functioning of the autonomic and somatic nervous systems, people living with SCI suffer progressive secondary health complications to the cardiopulmonary and musculoskeletal systems. While not comprehensive, this section provides a brief overview of some of the most common chronic health complications encountered by those with SCI.

Muscle atrophy: Disuse muscle atrophy is rapid and extensive, and occurs soon after injury. A transformation in the composition of the muscle is generally observed, where there is propensity towards an increased number of fast-twitch, Type II, highly fatiguable muscle

fibers in the muscle composition [11]. An increased proportion, or a shift in muscle fiber behavior, toward the Type II phenotype, generally results in a muscle with decreased fatigue resistance. Decreased fatigue resistance limits the ability to participate in prolonged periods of physical activity or exercise. This can therefore directly impact upon the magnitude of the response to rehabilitation activities such as Functional Electrical Stimulation (FES)-assisted leg cycle ergometry, Whole Body Vibration (WBV) training or robotics-assisted tilt-table or treadmill exercise. The reduction in muscle mass results in an increased susceptibility to skin complications and pressure sores due to a reduction in protection of the skin and subcutaneous tissue from pressure from the underlying skeleton. In addition, it has been suggested that the bone demineralisation is accelerated due to a reduced load from the muscle acting on the skeleton [12]. The sudden influx of calcium and protein to the body due to demineralisation and muscle atrophy can lead also to kidney problems in the early stages of cord injury [11].

Osteoporosis: Significant bone loss is reported in the first two years of SCI [13, 14]. The ‘mechanostat’ theory describes how the absence of normal bone loading from regular physiological activity leads to a rapid reduction in Bone Mineral Density (BMD) and bone strength [15], and therefore an increased risk of fracture. In some instances fractures can occur from a relatively minor trauma. Bone demineralisation causes a rapid influx of calcium which can later lead to hypercalciuria, ureteral stones or bladder stones [11, 16].

In severe cases, the intensive use of weakened bones during exercise may be avoided to minimise the risk of fracture. The treatment using a cast is generally avoided due to an increased risk of developing pressure sores (see below). Internal fixation is generally preferred, but carries the risks of surgery and infection [17]. It has been hypothesised that muscle activity plays a central role in the maintenance of skeletal BMD [12].

Pressure sores: A pressure sore, alternatively described as a bed sore or decubitus ulcer, occurs as a result of prolonged, unrelieved pressure or shear force, leading the formation of an ulcer and eventually an open wound. Pressure sores can lead to rehospitalisation, and are the primary cause for the re-admission of SCI patients in Scotland [3]. Surgical intervention and lengthy recovery periods are generally required. Pressure sores generally arise during extended periods of bed rest or wheelchair use, and are typically found over a bony prominence such as the ischial tuberosity, greater trochanter or sacrum. Increased pressure from underlying skeletal structures results in an insufficient supply of oxygen to the surrounding skin, subcutaneous fat and muscle, causing it to break down and become necrotic. If left untreated, sores can lead to infection and death. Pressure sores are particularly common in SCI, where compromised sensation, lack of mobility and muscle atrophy leads to an increased likelihood of their development [9, 10].

Orthostatic hypotension: Orthostatic Hypotension (OH), otherwise described as postural hypotension, is caused by the peripheral pooling of blood due to gravity. Vasodilation occurs during verticalisation, promoting circulatory collapse by compromising cardiac output and venous return. Symptoms typically include lightheadedness, dizziness, blurred vision, weakness, fatigue, cognitive impairment, nausea, heart palpitations (irregular heart beat), headache and syncope [18]. OH affects approximately 74% of early-stage SCI patients

during mobilisation, and prevents almost one third of these patients from participating in rehabilitation activities [19]. SCI patients are particularly susceptible to OH due to poor or absent vasomotor control arising which result from compromised autonomic function, and an ineffective ‘skeletal muscle pump’ due to muscle paralysis. OH is normally treated using tilt-table therapy, compressions stockings or an abdominal binder [9].

Spasticity: A commonly used definition of spasticity is ‘a motor disorder characterised by velocity dependant increase in tonic stretch reflexes with exaggerated tendon jerks, resulting from hyperexcitability of the stretch reflex, as one component of the upper motoneuron syndrome’ [20]. A spasm is an involuntary contraction of skeletal muscle and generally describes any condition of increased muscle tone [10], and occurs exclusively in those with Upper Motor Neuron (UMN) lesions. As such, the terms UMN lesion and spastic paralysis are used interchangeably.

Spasticity can be aggravated by irritative lesions, anal fissures, in-growing toe nails, pressure sores, fractures and urinary tract infections. Severe spasticity can hamper mobilisation, and strong spasms of the hip flexors can throw a patient from a wheelchair or fracture the femoral head [9]. A spastic imbalance of antagonistic muscles may result in hypertonia and contracture. Another form of spasm is clonus, or a clonic spasm, which is a rhythmic oscillation of a joint caused by a repetitive spasm in a particular muscle [10].

Autonomic dysreflexia: A noxious stimulus applied below the level of lesion can stimulate sympathetic outflow and elicit a vasoconstrictor reflex, hypertension and bradycardia (reduced heart rate). Peripheral vasodilation should normally occur to counteract these symptoms, but does not during autonomic dysreflexia because parasympathetic function is compromised in some cases of SCI. Appropriate corrective stimuli cannot pass distally through the injured spinal cord [21]. Autonomic dysreflexia is characterised by headache, sweating, and flushing of the skin. Without treatment it can lead to intracranial hemorrhage and death [9]. It typically arises due to a distended bladder but can also be caused by a pressure sore or FES. Those with a SCI at T6 or above are at the highest risk of suffering from autonomic dysreflexia.

1.2.5 Early Stage Spinal Cord Injury

A cycle of decline in general health subsequent to SCI is common and a sample pathway of this decline is shown in Figure 1.6. Immobilisation causes an initial decline in cardiopulmonary and vascular fitness due to inactivity. OH, caused principally by the SCI but contributed to by the lack activity also, delays participation in rehabilitation activities. Neuromuscular degeneration, particularly that of the neuromuscular junction and supporting structures causes a decline in muscle mass and function (muscle atrophy), minimising the ability to carry out intensive forms of exercise. The inability to exploit the cardiovascular demand of exercise, particularly the large demand exerted during the intense use of highly oxidative leg muscles, compounds the decline in cardiopulmonary fitness and an increases the risk of coronary heart disease and cardiovascular complications.

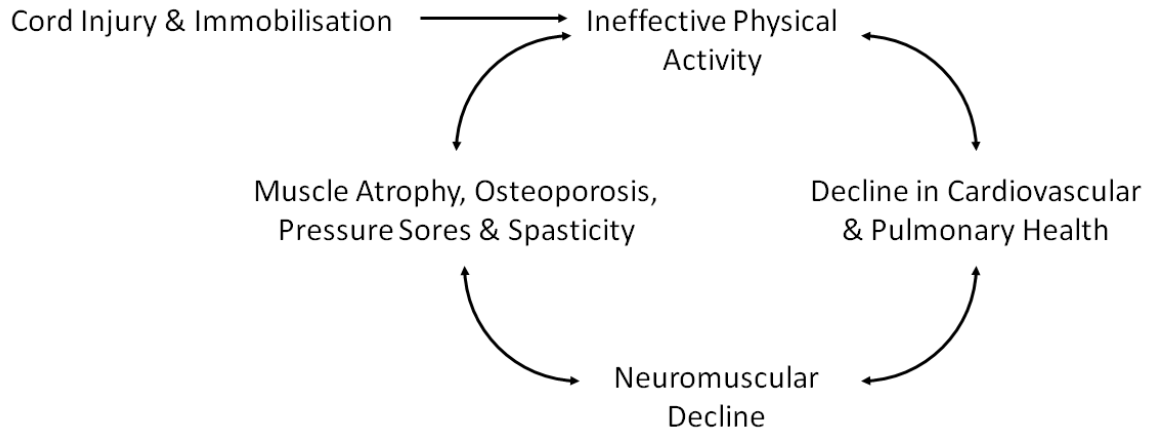


Figure 1.6: Summary of health cycle during early-stage SCI.

Two key elements were highlighted in this pathway. The first was the chronicity of injury: paralysis and inactivity result in muscle atrophy and reduces orthostatic tolerance, inactivity causes a decline in cardiopulmonary and vascular health, orthostatic hypotension delays participation in rehabilitation activities and contributes to muscle atrophy further. Degeneration of the neuromuscular junction and supporting structures accompanied by a general shift in the properties of the muscle fiber towards the Type II phenotype results in decreased fatigue resistance and an inability to carry out prolonged and intensive periods of exercise thus contributing to a further decline in cardiopulmonary and vascular health. A subcomponent of this pathway is the inability to use paralysed muscle. In the event that muscle is paralysed or cannot be utilised to its full effect, as is commonly the case in SCI, then the options available for carrying out exercise are limited. Furthermore, what exercise is carried out using paralysed muscle it typically of minimal benefit. In addition, it has been hypothesised that the reduced load exerted by paralysed muscles on skeletal structures leads to osteoporosis, and muscle atrophy increases the risk of pressure sores.

There is potentially a large benefit for any new exercise, rehabilitation protocol, or technology that can break this cycle of decline at the earliest possible time point after injury, before the rapid onset of declining health and fitness. The benefit would significantly increase the quality of life of the patient, accelerate their rehabilitation and reduce their cost of care.

1.3 Early Stage Rehabilitation

The principal focus of this research was to identify rehabilitation technologies that could be implemented during early stage SCI, and to assess their feasibility, with the specific goal of using the leg muscles to either: counteract neuromuscular degeneration, or attenuate the decline in cardiopulmonary or vascular health. Each of these factors were identified as key contributors to the cycle of decline which occurs during the early stages of injury (Figure 1.6). Two new rehabilitation technologies were identified for this purpose: Robotics-Assisted Tilt-Table Therapy (RATTT) and WBV. RATTT was investigated to determine its potential application for attenuating cardiopulmonary decline, and WBV was identified as a potential technology for counteracting neuromuscular degeneration. The specific aims of the

investigations into these technologies is discussed in greater detail below.

1.3.1 Tilt-Table

RATTT has primarily been used to counteract orthostatic hypotension in stroke, SCI and Multiple Sclerosis (MS) patients by activating the skeletal muscle pump using robotic orthoses attached to the legs. The requirement for orthostatic stability is therefore precluded and a patient may begin rehabilitation at an earlier time point. It was hypothesised that in addition to the therapeutic benefit of counteracting OH, that it may be used for exercise at the same time. Volitional effort during robotic assisted stepping on a treadmill and FES assisted cycling have been shown to result in increases in cardiopulmonary fitness in SCI populations. In each instance however, orthostatic stability was a requirement before starting these types of exercise. It was concluded that RATTT could potentially be used for the same purpose but with the added benefit of being employed at an earlier time point.

1.3.2 Vibration

Vibration has been used for various forms of therapy in the past, from slowly oscillating beds used to impart cardiopulmonary and vascular benefits on the elderly [22], hand held devices used to apply vibration directly to the muscle belly or tendon and therefore elicit a contraction, to passively driven, mechanical vibration systems which were designed to maintain bone mass during weightlessness in space flight [23]. A modern motorised version of this system, called WBV, is now available and is offered as a low impact, low cost alternative to conventional strength training and fitness programmes in neurologically intact-populations.

A participant stands on a vibration platform during WBV and the vibration is transmitted to the skeleton via the feet. The vibration is therefore indirectly imparted upon the neuromuscular system via the skeleton causing a muscle contraction. The response of paralysed muscle to WBV is poorly understood and has not been properly quantified. In the event that WBV does elicit a contraction in paralysed muscle then it may potentially be of therapeutic benefit, as there are a few other alternatives for muscle training (e.g. FES). WBV may be used to encourage muscle utilization during the early stages of SCI and allow the maintenance of muscle mass until such a time that intensive rehabilitation activities can begin. It was therefore hypothesised that a vibration induced contraction in paralysed muscle may be used to counteract neuromuscular decline and reduce the severity of spasm during early-stage SCI.

1.3.3 Aims & Objectives

The scope of this thesis was to focus on rehabilitation technologies that may be employed during early-stage SCI rehabilitation. The aim was to investigate the feasibility of these technologies and determine if they could potentially be used to disrupt the rapid cycle of decline that occurs soon after injury, specifically by attenuating the loss in cardiopulmonary and vascular fitness and muscle atrophy. The objectives were as follows:

- Determine the feasibility of RATTT as a form of exercise during early-stage SCI by measuring the acute vascular, pulmonary and ventilatory responses of an early-stage

SCI patient population during RATTT training. Assess if the training could be of sufficient intensity to potentially attenuate the decline in fitness.

- Develop a rehabilitation system that allows those with an early-stage SCI to stand on a WBV platform. Measure and quantify the magnitude of the acute neuromuscular response of a neurologically intact and SCI population to WBV in order to determine the feasibility of the system for early-stage SCI rehabilitation. Assess if the response was of sufficient magnitude to attenuate or counteract muscle atrophy.

Chapter 2

Robotic Assisted Tilt Table Therapy

SCI compromises motor function and sensation below the level of injury, resulting in paralysis and inactivity leading to reduced cardiopulmonary fitness and an increased risk of coronary heart disease and cardiovascular attack. These risks may be minimized through regular physical activity beginning at the earliest possible time point after injury, before extensive neuromuscular degeneration has occurred. RATTT may be used during this period for early-stage rehabilitation before orthostatic stability has been achieved. This chapter describes a cross-sectional, pilot study investigating the physical exertion associated with RATTT in SCI by measuring the acute responses of early-stage (<1yr) Motor Complete Spinal Cord Injury (CSCI) and Motor Incomplete Spinal Cord Injury (ISCI) patients to passive, active and FES-assisted robotic stepping. Active participation resulted in an increased response from the ISCI participants but the addition of FES did not consistently induce additional benefit. Extensive muscle atrophy was found to have occurred in those patients with CSCI, despite being recruited during the early stages of rehabilitation, and the effectiveness of the FES was therefore minimal. It was concluded that active participation in RATTT may be used to improve cardiopulmonary fitness in ISCI patients if implemented as part of a regular training programme. This research was published in the Journal of Rehabilitation Research and Development in 2013 [24].

2.1 Introduction

Physical inactivity is associated with obesity, diabetes and hypertension, and is a significant risk for the development of coronary heart disease and cardiovascular attack [7]. People with SCI suffer neurological impairment, which can result in severe disability and progressive secondary health complications to the cardiopulmonary and musculoskeletal systems. Their life expectancy is less than that of the general population [25], and they are at a higher risk of rehospitalisation and premature death due to cardiovascular disease and diabetes mellitus [26–30]. Rehospitalisation results in a considerable burden on the health-care system, emotional trauma and reduced quality of life [29, 31]. They lead sedentary lifestyles as a consequence of their injury, and their functional capabilities are less than those of their counterparts in the general population.

The risk of development of the health complications outlined above may be reduced through planned exercise and activity in the normal population [32]. The American College of Sports Medicine (ACSM) now recommend that the specific aim of cardiovascular training in the SCI population should be the same as that in the general population with no neurological deficit, and it should be to improve aerobic fitness [33]. Recommendations from the ACSM for aerobic exercise are consistent with the World Health Organisation (WHO), who advise that exercise activities should be between 3 - 6 Metabolic Equivalent (MET) carried out for 75 - 150min per week, depending on exercise intensity [34]. However, a joint report from the ACSM and Heart Association reduced the advised training intensity to between 50 - 85% maximum Oxygen Uptake (VO) for an elderly population due to the reduction in physical ability and the increased risk of harm from strenuous exercise, associated with ageing population [35]. The guidelines for the elderly are more appropriate during the early-stage SCI where physical disability and de-conditioning significantly compromise the ability to undertake and perform strenuous physical exercise.

Exercise capacity and functional ability in SCI is related to the neurological level and severity of cord injury [7, 36]. Muscle paralysis causes rapid and extensive muscle atrophy, which reduces daily energy requirements and leads to a decline in aerobic fitness. The inability to use highly oxidative muscles, such as those in the lower limb, reduces the ability to achieve a high metabolic demand during exercise. In addition, individuals with SCI have been found to have a diminished response to exercise when compared to the normal population, due to compromised autonomic function and poor or absent motor control. Hypokinesia [37], hypotension, reduced cardiac output [38], blunted blood pressure [39], and slow oxygen uptake kinetics [40] also occur during exercise.

Arm crank ergometry is the most commonly found and readily accessible form of exercise available to those with a SCI. Peak power outputs between 10 - 100W can be achieved during arm crank ergometry, depending on the neurological level and severity of injury [7]. The low metabolic demand of the relatively small muscle mass of upper limb, and the increased risk of shoulder pain [41] limits its effectiveness and use. Peak oxygen uptake levels achieved by healthy, neurologically intact test subjects during arm crank ergometry has been found to be 70% of that achieved during maximal treadmill testing [7]. However, unloaded static-FES training of the lower limb in a chronic SCI patient population has been shown to increase peak VO and heart rate [42]. These improvements were also shown to be consistent with a reduction in spasticity as determined using the Spinal Cord Assessment Tool for Spastic Reflexes (SCATS) measurement [43]. Other assistive technologies, that facilitate exercise using the lower limb, include robotics-assisted treadmill exercise and FES-assisted leg-cycle ergometry. Active participation during robotics-assisted treadmill exercise has been found to elicit an increased cardiovascular response from SCI subjects [44], and FES-assisted leg-cycle ergometry has been shown to improve cardiovascular fitness and exercise tolerance [40, 45, 46]. Peak power outputs in untrained people with SCI have been found to be between 10 - 13W during FES-assisted leg-cycle ergometry [47-49], and 12 - 48W during robotics-assisted treadmill exercise [44]. These technologies are typically employed in the chronic period of SCI, at a time point after which the individual has been accustomed to sitting and/or standing, and orthostatic tolerance has been achieved.

Tilt-table therapy is commonly used to treat orthostatic hypotension. During tilt-table therapy, an individual will be gradually accustomed to head-up-tilt by incrementally increasing tilt angle over a period of time. It has been found that tilt-table therapy can be enhanced by the application of FES to the knee extensors and plantar flexors. The improved orthostatic tolerance found was attributed to increased peripheral resistance caused by the muscle contractions elicited by FES [50]. Similar benefits have been found for healthy adults [51] and tetraplegics [52] using RATTT. RATTT integrates a standard tilt-table with robotic orthoses, that passively move the lower limbs in a manner that approaches normal, physiological hip kinematics. This passive movement has been shown to be effective in maintaining cardiovascular stability during head-up-tilt [51]. In addition, a study investigating RATTT augmented by FES concluded that the combination of these therapies was more effective in improving orthostatic tolerance than either intervention individually [53, 54].

In addition to improving orthostatic tolerance, RATTT may also be an effective exercise tool. Initiating the rehabilitation process at the earliest possible time point may minimise losses in aerobic fitness that arise due to inactivity in the early-stages of SCI. It was hypothesised that by employing RATTT in this way, the dual purpose of increasing orthostatic tolerance and attenuating the decline in aerobic fitness may be achieved.

2.2 Aims & Objectives

The metabolic demand exerted during passive, active and FES-assisted stepping during RATTT has not yet been investigated in the SCI patient population. The aim of this study was two-fold: to investigate the physical exertion of RATTT, by describing the ventilatory and cardiopulmonary response to passive, active and FES-assisted RATTT; and to investigate the difference in response of ISCI and CSCI patients. This was done by addressing by the following technical and physiological objectives.

2.2.1 Technical Objectives

The technical objectives were:

1. Develop a visual feedback system to encourage greater volitional participation during robotic assisted stepping.

2.2.2 Physiological Objectives

The physiological objectives were:

1. Quantify the ventilatory and vascular responses of ISCI and CSCI participants during different types of RATTT exercises.
2. Determine the difference responses between ISCI and CSCI participants.

2.3 Experimental Methods

2.3.1 Subjects

Neurologically and orthopedically stable, non-acute (> 1 month) SCI patients were recruited from the Queen Elizabeth National Spinal Injuries Unit for Scotland: three CSCI and three ISCI. The main inclusion criteria were patients with paraplegia or tetraplegia due to a spinal cord lesion with no lower motor neuron damage. The study was approved by the NHS South Glasgow and Clyde Local Research Ethics Committee (ref: 08/S0710/66). Each subject provided informed consent prior to participation.

Table 2.1: Subject characteristics. AIS: American spinal injuries association impairment scale; CSCI: Motor complete SCI; ISCI: Motor incomplete SCI; SCI: Spinal Cord Injury; SD: Standard deviation; M: Male.

Subject	Lesion	Level	Gender [yrs]	Age [kg]	Weight [wks]	Time post-injury	AIS
1	CSCI	T4	M	22	70	16	A
2	CSCI	T8	M	18	76	18	A
3	CSCI	C3	M	25	60	21	B
Mean (SD)	-	-	-	22 (4)	69 (8)	18 (3)	-
4	ISCI	T9	M	53	86	46	C
5	ISCI	T2	M	44	98	40	D
6	ISCI	C4	M	54	81	9	C
Mean (SD)	-	-	-	50 (6)	88 (9)	32 (20)	-

2.3.2 Robotics-assisted tilt-table

The RATTT device (Erigo, Hocoma AG, Switzerland), shown in Figure 2.1, utilises a tilt-table with integrated linearly driven, lower limb robotic orthoses. The table can be tilted from $0 - 80^\circ$ from horizontal. In addition, the table was actuated at the hip and allowed hip extension up to 20° . The robotic orthoses were attached to the thighs and impose a stepping profile on the lower limbs. An upper body harness secured the participant to the table and provided support around the chest and abdomen. The feet were secured to foot plates which had an integrated spring system. Each spring provided a resistance up to 450N at maximum extension, and stepping rates ranged from 4 to 40 steps/minute/leg. The power required to extend both integrated springs fully was 15W (Equation 2.1) at the highest stepping rate.

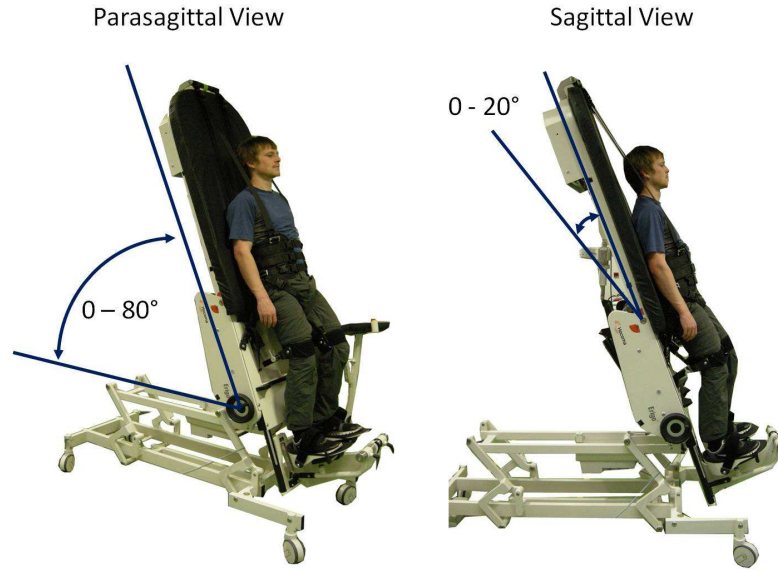


Figure 2.1: Tilt-table with integrated robotics-assisted stepping device shown in a sagittal and parasagittal view.

$$W = \frac{F \cdot x \cdot \dot{s}}{2} \quad (2.1)$$

Where W = work; F = spring force; x = spring extension; and \dot{s} = stepping rate. Maximum spring extension was 0.05m.

2.3.3 FES

FES is a method by which an electrical impulse is applied to a peripheral nerve bundle. Among several applications, FES may be used for therapeutic effects, such as the relief of pain or reduction of spasm [43]; in the study of neural pathways and reflexes [55], such as the H-reflex; or to produce function or movement, such as FES-Leg Cycle Ergometry [45]. In the context of this thesis, the application of FES to produce function in the form of limb movement is used. In this context, FES was described by Rushton as generating “*movements or functions which mimic normal voluntary movements, and so to restore the functions which those movements serve*” [56]. FES was used in this study to restore the function of stepping in the paralysed limb during RATTT.

FES was applied during stepping using a bi-phasic, current controlled electrical stimulation device (Motionstim, Krauth+Timmermann, Germany). Stimulation was delivered by transcutaneous neuromuscular electrical stimulation electrodes (PALS Platinum, Axelgaard, Denmark) placed over the quadriceps ($5 \times 10\text{cm}$ oval), hamstring ($5 \times 10\text{cm}$ oval) and calf ($5 \times 6.4\text{cm}$ oval) muscle groups. Stimulation frequency and pulse width were fixed at 20Hz and $300\mu\text{s}$ respectively. Stimulation current was adjustable for each individual muscle group. Stimulation timing was automatically synchronised with the RATTT device during stepping: the hamstring and calf muscle groups were stimulated during knee flexion and the quadriceps muscle group was stimulated during knee extension.

2.3.4 Feedback System

Hip position was measured from an integrated position sensor on the RATTT device and fed back to the patient to encourage greater participation during stepping. The hip position signal was acquired in real-time at 10Hz using a 12bit USB analog-to-digital converter (NI USB-6008, National Instruments Corporation, Texas, USA). Data acquisition was synchronised and displayed using LabVIEW 8.6 (National Instruments Corporation, Texas, USA). An example of the basic graphic displayed in real-time to the participant is shown in Figure 2.2.

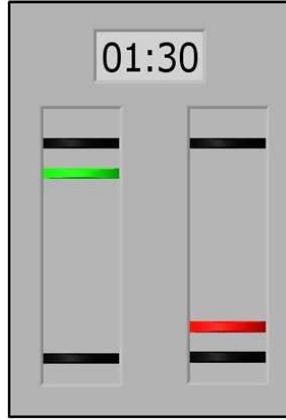


Figure 2.2: Screen shot of RATTT real time position display

The graphic showed hip position normalised to maximum hip flexion and displayed throughout each step. The extremes of hip movement were shown as black bands and pre-determined limits were set within these extremes for each participant. The participant was instructed to achieve and exceed the pre-determined limits, where possible, in order to encourage greater participation in the stepping exercise. The real time position display would change in colour from red to green if the limits were exceeded. The limits were manually adjusted throughout a testing session if the participant consistently exceeded or missed the limits.

2.3.5 Measurement equipment

Pulmonary gas exchange and ventilatory measurements were performed using a breath-by-breath analysis system (MetaMax 3B, Cortex Biophysic GmbH, Germany) and recorded on a laptop PC. Prior to use, the system was calibrated by performing a volume analysis, using a 3 litre volumetric syringe, and gas calibration was carried out using ambient air and a certified precision-analysed gas mixture. Heart rate was measured and recorded using a short range telemetry heart rate monitor (Polar S410, Polar Electro Oy, Finland). Blood pressure measurements were performed using an automatic blood pressure monitoring device (M7, Omron Healthcare Europe B.V., The Netherlands).

2.3.6 Testing protocol

There were three experimental sessions per participant. During the first session, the participant was familiarised with the RATTT device and all measurement equipment. Testing was carried out in the two subsequent experimental sessions. The participant was asked not

to perform any strenuous exercise, or consume alcohol, in the 24 hours prior to testing, or to ingest any caffeine in the 4 hours, or food in the 2 hours, prior to a testing session. No two experimental sessions were carried out on consecutive days. The participant wore similar clothing and was in good health for each session.

The participant was fitted with the heart rate monitor and secured to the RATTT device using the upper body harness at the beginning of each experimental session. Transcutaneous neuromuscular electrical stimulation electrodes were then placed over the quadriceps, hamstring and calf muscle groups while in a supine position. Stimulation was then applied and the current was increased (28 - 100mA) for each individual muscle group to the point where a palpable muscle contraction was detected. The participants legs were then secured to the linearly driven orthoses and foot plates. A cuff was then placed above the elbow of the left arm to allow blood pressure measurement from the brachial artery. The participant finally donned the mask for the breath-by-breath analysis system.

Each testing session consisted of five discrete phases of 5 minutes duration. Table 2.2 describes each discrete test and the order in which it was carried out.

Table 2.2: RATTT testing protocol. ISCI = Motor incomplete spinal cord injury. CSCI: Motor complete spinal cord injury.

Phase	Participant	Discrete Test
1	CSCI & ISCI	Supine posture and with no stepping.
2	CSCI & ISCI	Titled to an angle of 70° from horizontal.
3	CSCI & ISCI	Robotic orthoses activated.
4a	ISCI	Volitional participation during stepping based on visual feedback.
4b	CSCI & ISCI	FES applied to augment to participation in the stepping cycle.

The participants worked against the spring resistance system during knee extension, and the ISCI participants continued to volitionally participate in the stepping cycle during to the application of FES in Phase 4b. Pulmonary gas exchange, ventilatory and heart rate measurements were continuously recorded throughout the course of a testing session.

Discrete blood pressure measurements were taken in the final 30s of each phase. Stepping rate was set and fixed at a value between 20 - 40 steps/minute. At these rates, the estimated power required to extend the integrated spring system was between 3.75 - 7.5W. Hip extension was fixed at 0° throughout the experimental session.

2.3.7 Outcome Measures

The technical outcome measures directly associated with these methods were:

1. Hip Position - hip position was measured in real time from the RATTT device and used to provide real-time feedback to the participant.

The physiological outcome measures were:

1. Oxygen Uptake - measured directly from each participant and normalised to body weight.

2. Respiratory Exchange Ratio - calculated from oxygen uptake and carbon dioxide output.
3. Heart Rate - measured directly from the participant and normalised to the target heart rate.
4. Mean Arterial Blood Pressure - calculated from the discrete blood pressure measurements taken at the end of each discrete test.

2.4 Analysis Methods

Data processing was performed using the Matlab Signal Processing Toolbox (Matlab R2010a, The Mathworks Inc., USA). Pulmonary, ventilatory and heart rate data were smoothed using a 10th order, moving average filter. A 10th order moving average filter was implemented to minimise the high frequency, natural fluctuation in oxygen uptake, breathing rate and heart rate observed on a breath-by-breath basis and thus allow for the underlying changes in these parameters to be observed as each phase of testing progressed. The maximum value in the final 90s of each phase was then found. Each outcome measure was averaged, for each subject respectively, between the first and second testing sessions.

2.4.1 Equations

Heart rate was normalised to each participants own target heart rate using equation (2.2) [1,7]:

$$HR_N = \frac{HR}{220 - age} \quad (2.2)$$

Where, HR_N : Normalised heart rate. HR : Recorded heart rate.

Mean arterial blood pressure was calculated using equation (2.3) [57]:

$$MAP = \frac{P_{sys} - P_{dia}}{3} + P_{dia} \quad (2.3)$$

Where, P_{sys} : Systolic blood pressure. P_{dia} : Diastolic blood pressure.

2.4.2 Statistical Analysis

Statistical analysis was performed using the Matlab Statistics Toolbox (Matlab R2010a, The Mathworks Inc., USA). Data normality was assessed using quantile-quantile plots and Levene's test was used to test for equal variance. A repeated measures Analysis of Variance (ANOVA), and post-hoc multiple comparisons procedure with Bonferroni correction, was used to investigate the response, of the ISCI and CSCI subjects respectively, to each phase of the testing protocol. Paired t-tests were used to investigate the difference in response of the ISCI and CSCI subjects for a given phase of testing. Pearson's correlation coefficient was used to investigate the test-retest reliability of the obtained outcome measures [58]. Cohen's d, based on an average standard deviation, was used to estimate effect size.

2.5 Results

All participants completed each of the experimental sessions. Head-up-tilt was tolerated well and no instances of hypotension or autonomic dysreflexia occurred. The oxygen uptake, respiratory exchange ratio, minute ventilation and heart rate response of each of the tested subjects are shown in Figure 2.3.

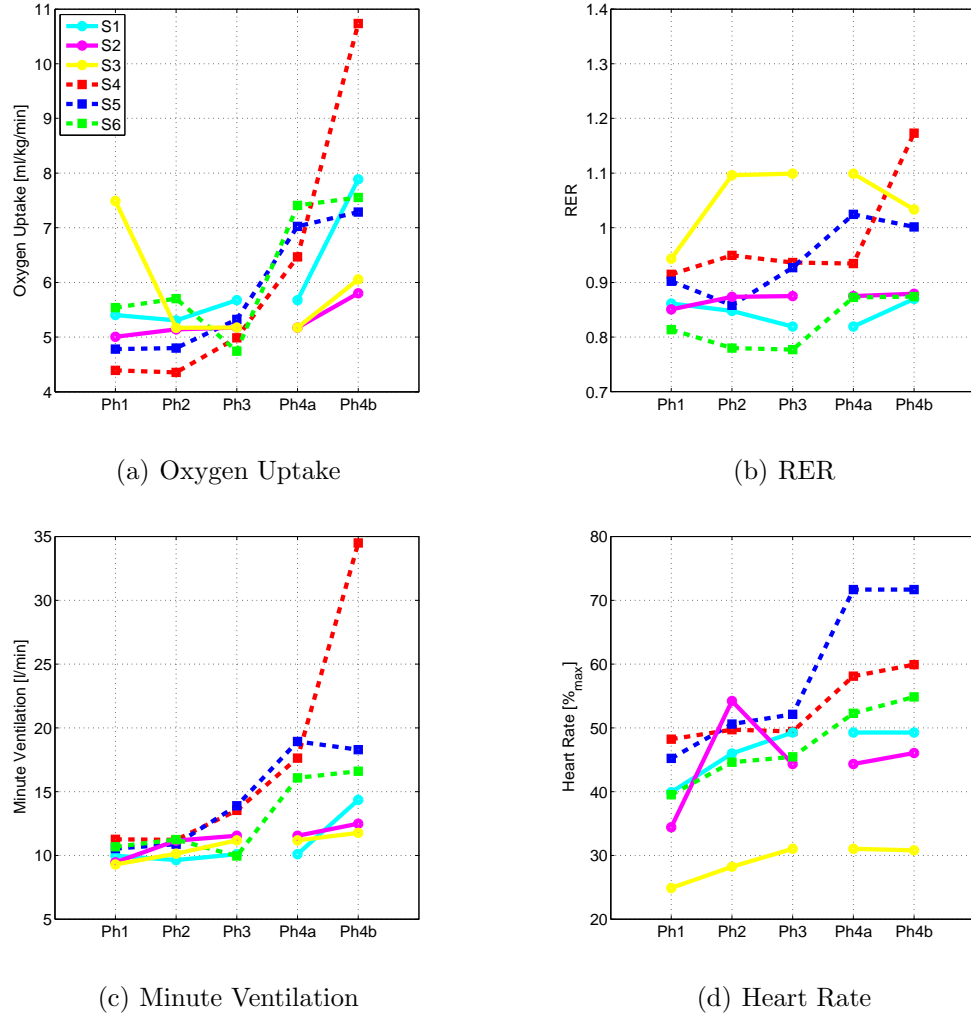


Figure 2.3: Plot of individual results. Ph1: Supine posture with no stepping; Ph2: Titled to an angle of 70° from horizontal; Ph3: Robotic orthoses activated; Ph4a: Volitional participation during stepping based on visual feedback (ISCI only); Ph4b: FES applied to augment to participation in the stepping cycle; ISCI: Incomplete spinal cord injury; CSCI: Complete spinal cord injury.

For the ISCI subjects, it was found that oxygen uptake, respiratory exchange ratio, minute ventilation and heart rate were unchanged throughout the first three phases (Phase 1 - 3) of the testing protocol (see Table 2.3). Volitional participation (Phase 4a) in the stepping cycle and the addition of FES (Phase 4b), led to an increase in oxygen uptake, respiratory exchange ratio, minute ventilation and heart rate, and these increases were found to be significant. No statistically significant change in mean arterial blood pressure was found throughout the testing protocol. For the CSCI subjects, a small and statistically significant

increase in minute ventilation was identified, but no change in the other outcome measures was found. The results of the statistical analysis are summarised in Table 2.3.

Pearson's correlation coefficient indicates a high degree of test-retest reliability for both the ISCI and CSCI subjects. Correlation coefficients were found to be between 0.64 - 0.95, and were significant at $p < 0.05$, implying that there is a large or very large between test relationship for each of the outcome measures.

In comparing the ISCI and CSCI subjects, it was found that there was no significant difference between either groups oxygen uptake, respiratory exchange ratio, minute ventilation or heart rate response throughout the first three phases (Phase 1 - 3) of testing. During Phase 4b, it was found that the oxygen uptake, minute ventilation and heart rate of the ISCI subjects were significantly larger than those of the CSCI subjects in the same phase. The significantly larger responses found for the ISCI subjects is attributed to the large effect, as computed using Cohen's d , of volitional participation during this phase. Cohen's d was calculated to be 1.32 (VO), 1.49 (Minute Ventilation (MinVen)) and 2.22 (Heart Rate (HR)) respectively. However, despite the overall group averages for the ISCI subjects being significantly larger than those for the CSCI subjects, a review of the the individual changes on a case by case basis indicates that the increases in the ISCI group were principally attributable to subject S4. This is most likely due to the fact that subject S4 was less affected by the severity of cord injury (T9) compared to the other ISCI participants (T2 & C4). This is discussed in greater detail in the following section. Mean arterial blood pressure was found to be significantly larger across all phases for ISCI subjects when compared to the CSCI subjects.

Table 2.3: The mean oxygen uptake, RER, minute ventilation, heart rate and mean arterial blood pressure, for the incomplete and complete spinal cord injured subjects respectively, are presented for each phase of the experimental protocol. SD: Standard deviation. ISCI: Motor incomplete spinal cord injury. CSCI: Motor complete spinal cord injury. F: Repeated measures ANOVA F-statistic. DF: Degrees of freedom. p: Significance of F-statistic.

Parameter	Injury	Phase 1 Mean (SD)	Phase 2 Mean (SD)	Phase 3 Mean (SD)	Phase 4 Mean (SD)	Phase 5 Mean (SD)	RM F(DF), p
Oxygen Uptake [ml/kg/min]	ISCI	4.90 (0.57)	4.95 (0.72)	5.02 (0.32)	6.97 (0.85)	8.53 (1.79)	F(4)=6.88,p=0.01
	CSCI	5.96 (1.35)	5.20 (0.57)	5.34 (0.28)	-	6.58 (1.06)	F(3)=1.48,p=0.31
RER	ISCI	0.88 (0.05)	0.86 (0.11)	0.88 (0.10)	0.94 (0.08)	1.02 (0.14)	F(4)=3.93,p=0.05
	CSCI	0.89 (0.06)	0.94 (0.13)	0.93 (0.14)	-	0.93 (0.09)	F(3)=0.84,p=0.52
Minute Ventilation [l/min]	ISCI	10.82 (0.64)	11.10 (0.67)	12.45 (2.33)	17.54 (3.01)	23.12 (9.65)	F(4)=4.41,p=0.04
	CSCI	9.57 (0.45)	10.31 (1.28)	10.93 (0.91)	-	12.86 (1.43)	F(3)=6.62,p=0.02
Heart Rate [% _{max}]	ISCI	44 (5)	48 (4)	49 (6)	61 (11)	63 (9)	F(4)=11.63,p<0.01
	CSCI	33 (7)	43 (13)	42 (9)	-	42 (9)	F(3)=3.96,p=0.07
Arterial Pressure [mmHg]	ISCI	104.17 (18.82)	108.17 (20.73)	118.17 (23.53)	110.67 (25.55)	114.83 (16.69)	F(4)=1.05,p=0.44
	CSCI	86.78 (2.47)	85.67 (10.98)	84.78 (13.45)	-	87.39 (13.34)	F(3)=0.08,p=0.97

2.6 Discussion

This cross-sectional, pilot study investigates the feasibility of RATTT as a potential exercise therapy. It is hypothesised that this technique may be employed during the very early stages of rehabilitation to facilitate cardiopulmonary exercise. It may be of most benefit during the acute stage of injury, before orthostatic tolerance is achieved and more traditional exercise techniques have commenced. The participants in this study were within their first year of injury and were recruited because they were orthopedically and neurologically stable. These subjects would be considered to be early-stage rather than acute patients.

In agreement with previous studies [53, 54], head-up-tilt was well tolerated during RATTT with no instances of pre-syncope or syncope occurring. Some patients, particularly those with inhibited motor function and compromised vasomotor control, would be considered to be at a high risk of orthostatic instability. When considered as a group, no significant changes in mean arterial blood pressure were found during any phase of the testing protocol for the incomplete or complete SCI subjects. The mean arterial blood pressure of the ISCI group was found to be significantly higher than that of the CSCI group. This is most likely a consequence of the higher average age of the incomplete SCI (50 years) compared to the complete SCI (22 years) group, where systolic and diastolic blood pressure are generally expected to increase with age [59]. Alternatively, those with an incomplete injury may be exhibiting increased venous return in comparison to their complete counterparts, due to the presence of an effective skeletal muscle pump, particularly during active movement of the lower limb. Increased venous return would result in higher mean arterial blood pressure as recorded at the brachial artery. Some short-duration spikes in mean arterial blood pressure may have occurred during the transition from one testing condition to another but the likelihood of such changes being detected is low because blood pressure was recorded only at discrete time intervals, which were at the end of each phase. Such spikes were found in the continuously recorded heart rate of two subjects (Subjects 1 & 2) at the beginning of head-up-tilt (Phase 2) and at the initiation of robotic assisted stepping (Phase 3). These sharp increases however, soon dropped and stabilised at the reported values.

When considered together, the responses of the ISCI and CSCI groups are generally comparable throughout the first three phases (Phase 1 - 3) of the testing. Small increases in minute ventilation, oxygen uptake and heart rate were found during head-up-tilt (Phase 2) and head-up-tilt with passive robotics assisted stepping (Phase 3). However, these increases were not found to be significant and no significant difference between the ISCI and CSCI groups were elicited.

Due to the nature of their paralysis, the CSCI subjects were unable to volitionally contribute to the robotics assisted stepping cycle (Phase 4a). Active participation during this phase by the ISCI subjects did elicit an increase in oxygen uptake, minute ventilation and heart rate and the increase in heart rate was found to be significant.

FES was used to augment the ISCI subjects' volitional contribution during the final phase of the testing protocol (Phase 4b). The CSCI participants progressed directly to this phase upon completing Phase 3. For the CSCI subjects, the increased cardiopulmonary and ventilatory

responses found due to FES were small, but significant in the case of minute ventilation. These increases however are unlikely to be of sufficient magnitude to lead to improved fitness if implemented in a training programme. Larger increases in these parameters were found for the ISCI subjects, and in most cases the increases were found to be significant. These results indicate that FES potentiates the cardiopulmonary and ventilatory response of ISCI subjects during RATTT. An inspection of the ISCI group averages reveals that volitional participation during FES-assisted RATTT results in the largest increases in cardiopulmonary and ventilatory response. Considering each subject individually though, analysis indicates that the continued increases found are attributable to Subject 4 only. The responses from Subject 5 & 6 remain consistent throughout the last 2 phases of testing (Phase 4a & 4b).

It is established that functional ability and response to exercise are related to neurological level and severity of cord injury [7]. Those with a lower level injury are expected to have more functional muscle available with which physical activity can be performed. Investigations comparing individuals with incomplete tetraplegia, high-paraplegia and low-paraplegia report that physical capacity for exercise decreases with increasing levels of cord injury [36]. Decreased capacity for exercise is manifested as a lower achievable peak oxygen uptake and lower peak heart rate. In this study, it is thus expected that those with a lower level injury will perform better.

In keeping with this assumption, it was found that the subject with low-paraplegia (Subject 4) performed considerably better than those subjects with higher level injuries. A review of the of the individual VO and minute ventilation parameters for subject S4 shows a rapid increase in each parameter with prolonged volitional participation. By extension, subject S5 would be considered to be less affected by the severity of cord injury (ASIA D & T6) than subject S6 (ASIA C & C4), but the VO and minute ventilation values were broadly comparable for each. Surprisingly, the peak heart rate recorded for subject S5 was considerably higher than either S4 or S6, and this heart rate was inconsistent with oxygen consumed. This is in contrast to S4 in particular, for whom all physiological parameters indicate was performing work at a much higher intensity. One possibility is that subject S6, who was 9 weeks post-injury was less affected by physical de-conditioning than subject S5, who was 40 weeks post-injury. Furthermore, subject S5 was considerably heavier (98kg) than subject S6 (81kg) and despite having a lower level and severity of injury, was likely to have been in poorer physical condition at the time of injury. The cord injury likely compounded issues associated with physical conditioning and thus negated any potential benefits in exercise performance associated with a lower level injury. It should be noted that such observations are not limited to SCI and those who are in poorer physical condition are unlikely to be able to perform sustained physical activity. Furthermore, those starting at a lower physical baseline generally associate a larger perceived rate of exertion with a lower level of exercise intensity when compared to those that are in better health. [35].

By contrast, the results from the CSCI subjects and intra-group comparisons thereof, are consistent with the level and severity of cord injury. Subjects S1 and S2 each had a comparable level and severity of injury, and each exhibited a larger response to the FES-assisted RATTT stepping exercise than subject S3. Somewhat surprisingly, subject S1 exhibited a moderately

larger response than subject S2, despite having a higher level cord-injury. The differing responses from each of these subjects principally occurred upon the application of FES. A review of available information on physical conditioning indicates that each was within the same age group and were approximately the same weight, and each was therefore likely to have been affected by physical de-conditioning to the same degree. However, it was noted that subject S1 suffered to a greater degree from spasm than subject S2. The extent of neuromuscular decline was therefore less and the physical exertion associated with subject S1 was therefore likely to be larger upon the application of FES, as was seen in this study.

Oxygen uptake and respiratory exchange ratio are key indicators of the stress being exerted on the cardiopulmonary system during exercise. The test-retest reliability of these parameters was found to be high, and as such they provide a reliable indication of the degree of physical exertion experienced during RATTT. Oxygen uptake is commonly expressed as a MET where 1 MET is equivalent to a normalised oxygen uptake value of 3.5 ml/kg/min, which is the approximate resting metabolic rate of a typical 70kg, 40 year old male [1]. For the ISCI subjects, normalised oxygen uptake was recorded as between 5.3 - 11.0 ml/kg/min during Phase 4 of the testing protocol, which equates to 1.5 - 3.1 MET. This is defined by the WHO to be of low to moderate intensity physical activity. Moderate-intensity physical activity is defined as being between 3 - 6 MET's and high-intensity physical activity is at 6 MET's and above [34]. The peak oxygen uptake values found in this study are considerably lower than the maximum achievable values in age matched persons from the general population, which are expected to be in the range of 34 - 44 ml/kg/min [60]. However, such levels of oxygen uptake are difficult to achieve in SCI persons due to an inability to effectively recruit all available muscle. A more appropriate comparison is peak oxygen uptake in chronic ISCI persons during wheelchair and leg cycle ergometry, where values of 11.14 - 21.6 ml/kg/min are typical [48, 61]. In summary, the levels of oxygen uptake elicited in the ISCI subjects during volitional participation in RATTT approach the peak values expected of this patient population. In addition, respiratory exchange ratio did not usually exceed 1, indicating that the intensity of the physical activity is aerobic in nature and is likely to be sustainable. These data suggest that volitional participation in RATTT may be used for low to moderate intensity physical activity, and if instituted regularly it is likely that this form of activity may be used to maintain cardiopulmonary fitness.

For the CSCI subjects, normalised oxygen uptake was found to be between 5.0 - 7.5 ml/kg/min during FES-assisted RATTT, which equates to a MET of 1.4 - 2.1. In comparison, maximum oxygen uptake in age matched individuals from the general population are expected to be between 43 - 52 ml/kg/min [60]. However, as with the ISCI subjects, these levels of oxygen uptake are unlikely to be attainable in those with SCI. The peak oxygen uptake values recorded in this study are similar to those reported for chronic, untrained CSCI individuals during FES-assisted leg cycle ergometry, where typical values were found to be 7.3 ± 2 ml/kg/min [45]. These comparisons are considered to be most valid because FES-assisted leg cycle ergometry, like RATTT utilises the lower limb only with little or no contribution from the upper limb. It is unlikely that FES assisted RATTT will result in improved cardiopulmonary fitness as it would be necessary to maintain this level of intensity of physical activity for a duration in excess of 150mins per week. Nonetheless, this type of

exercise may be sufficient to attenuate the loss in cardiopulmonary fitness during the acute stages of SCI.

When comparing the complete and incomplete SCI subjects during FES assisted RATTT (Phase 4b), it was found that oxygen uptake, minute ventilation and heart rate were significantly larger for the ISCI subjects. The computed effect size for each of these parameters was found to be large, indicating that the type of injury, and by extension the ability to volitionally contribute to the stepping cycle, largely accounts for the difference in response found. These data suggest that volitional participation is key in eliciting an increased cardiopulmonary and ventilatory response in SCI participants during RATTT.

This study assessed a small sample of SCI subjects. The average age, weight and time since injury of the CSCI group (22years, 67kg, 18weeks) was considerably different to that of the ISCI group (50years, 88kg, 32weeks). Extensive muscle atrophy was found to have occurred for the CSCI subjects at the time of recruitment, and it is established that persons with SCI can exhibit a slow response to exercise [40], and may have a blunted heart rate [27]. Fatigue resistance to exercise is compromised due to oxidative, Type I muscle fibers taking on the properties of the glycolytic, Type II muscle fibers [62]. These considerations affect the capacity of the aerobic systems to undertake and respond to physical activity. This is reflected by the fact that peak oxygen uptake levels recorded in this investigation are much lower than those potentially achievable in the general population, but are comparable to similarly untrained SCI persons during other forms of exercise.

2.7 Conclusions

A limitation of this study was the inability to perform a real-time measurement of the work being performed by the subject during RATTT. An estimate of the total power required to work against the integrated spring system of the RATTT was given, but it is not known what proportion of this work done was performed by the individual and the robotic orthoses respectively. Nonetheless, it was clear from changes in oxygen uptake, minute ventilation and heart rate that some degree of work was being performed as these parameters reflect an increased metabolic demand from the muscles as the prescribed exercise was carried out. Given this small and complex case mix, clear trends were shown. It was identified that volitional effort resulted in an increased cardiopulmonary and ventilatory response during RATTT, and these increases were sustained or marginally improved upon with the addition of FES. In this study, changes in minute ventilation, oxygen uptake, heart rate, and respiratory exchange ratio were each found to be significant for the incomplete SCI subjects. This evidence is sufficient to warrant further investigation with a larger sample population of SCI subjects with tighter control of age and time since injury to determine the dose response to a period of training. It was concluded that a period of RATTT training with volitional contribution could potentially improve cardiopulmonary and ventilatory fitness in those with an ISCI, and FES-assisted RATTT may be sufficient to attenuate losses in fitness in those individuals with CSCI.

Chapter 3

WBV Literature Review

Investigation into the therapeutic benefit of vibration therapy dates back to the 1930s. Slowly oscillating beds were initially shown to impart a cardiopulmonary and vascular benefits on elderly, immobilised patients [22]. The series of investigations which followed confirmed this and recovery post-bed rest was shown to be faster on an oscillating bed when compared to a non-oscillating bed [63]. Investigation into the neuromuscular effect of vibration began in the 1960s when hand held devices were used to apply vibration directly to the muscle belly or tendon. Electromyography and microneurography studies ultimately led to the characterisation of the Tonic Vibration Reflex (TVR) [64]. Interest into the therapeutic benefits of vibration therapy for the musculoskeletal system, particularly to that of the lower limb, has developed more recently and research into the field has risen steadily since the mid-1990s.

The first conventional form of vibration therapy was a passively driven, mechanical vibration system developed for the Euro-Mir 95 mission. The mechanical impact from the vibration system is analogous to the transient loading experienced at heel strike during walking. This mechanical stimulus was effective in maintaining bone mass during weightlessness in space flight [23]. Since Euro-Mir 95, interest has grown considerably in commercially available, motorised alternatives to this system. Vibration therapy is now commonly applied indirectly through the feet via an actuated vibration platform that supports the person, rather than via an oscillating bed, hand held device, or passively driven machine. Vibration therapy applied in this way is called Whole Body Vibration (WBV).

In addition to the broad array of other scientific investigations, a series of bed-rest studies were carried out between 2003 - 2007 investigating WBV. Bed-rest is now used as an ‘earth-based’ model of the physical de-conditioning that is associated with prolonged weightlessness during space flight. The potential application of WBV to counteract this decline was investigated in the bed-rest studies [65–71]. More recently, WBV training was incorporated into the Mars 500 mission, which was a collaboration between the European Space Agency and the Russian Institute of Biomedical Problems. The Mars 500 mission simulated 520 days of isolation which is the time expected to travel from Earth to Mars and back. The effect of WBV on cardiovascular and pulmonary function, muscle function, muscle force, muscle power, bone mass, and bone strength were investigated.

The growing interest in WBV is clear. It is offered as a low impact, low cost (time and economically) alternative to conventional strength training and fitness programmes. It is thus becoming attractive as an exercise modality not only for space flight, but for those in the general population, athletes, the elderly, and those with a neurological deficit such as: Spinal Cord Injury (SCI), stroke, and multiple sclerosis. This review presents the main theories which describe the neural mechanism by which WBV causes muscles to contract and then focuses on four of the key areas of WBV research, including investigations into: (i) the neuromuscular response to WBV as measured using EMG, (ii) the effect of WBV on the EMG recording itself, (iii) the physiological response to a recent bout of WBV and (iv) the physiological response to a prolonged period of WBV training.

3.1 Introduction

3.1.1 Vibration

Vibration is a periodic mechanical disturbance, that can be described by its amplitude (A) and frequency (f). In most applications, the shape of the mechanical disturbance is sinusoidal (Figure 3.1), and its peak acceleration (a_{peak}) can be calculated by Equation 3.1.

$$a_{peak} = \omega^2 A \quad (3.1)$$

Where angular frequency $\omega = 2\pi f$.

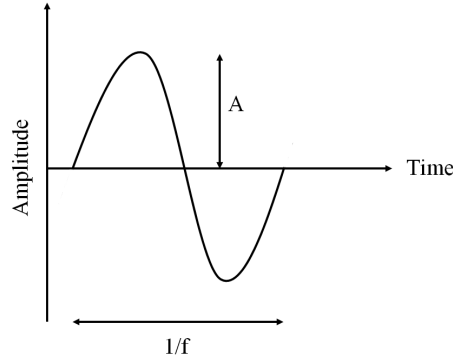


Figure 3.1: Shape of vibration displacement.

3.1.2 Vibration Device

Vibration can be applied directly to the muscle or tendon, or indirectly via a vibrating platform. Indirect vibration is more commonly known as WBV, and can be applied in a number of ways: Vertically-Vibrating (VV)-, Side-Alternating (SA)- or Rotating-WBV platforms. The VV- and SA-WBV platforms are most common, and are similar because the main component of displacement is in the vertical direction (Figure 3.2). During VV-WBV, displacement is exclusively in the z-direction, whereas during SA-WBV, the platform oscillates about its central axis, thereby resulting in a small component of displacement in the y-direction and the majority of displacement in the z-direction. During Rotating-WBV, the platform is displaced about its centre point, with the main components of displacement

in the x- and y-directions. There is little evidence in the literature describing Rotating-WBV or advocating its use, and it is therefore not described any further in this thesis.

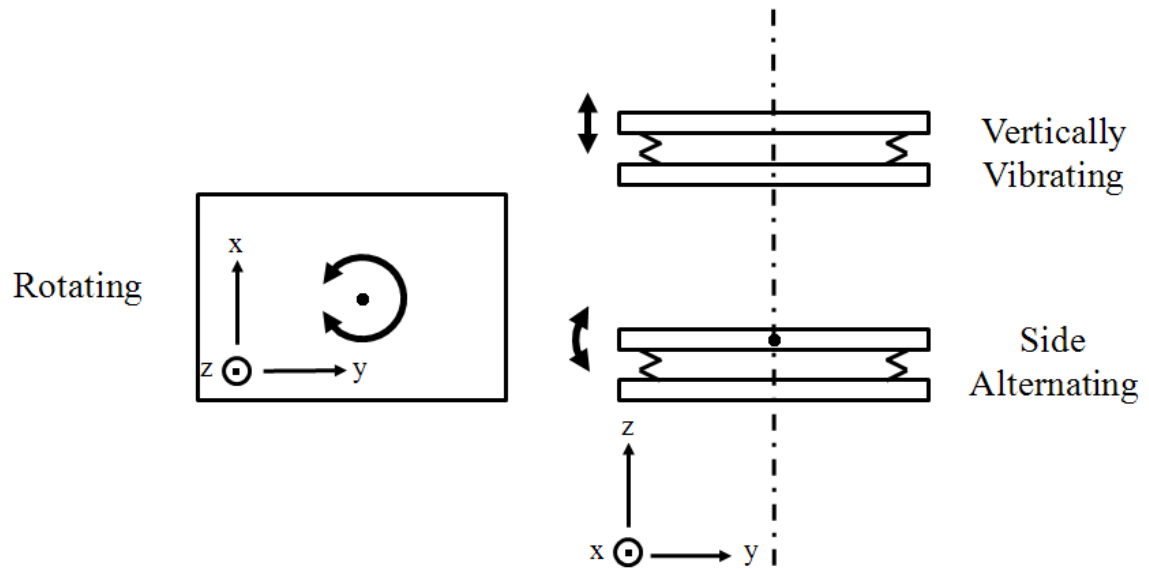


Figure 3.2: Schematic comparing different whole body vibration platforms.

Vibration is transferred through the feet to the skeleton and tendons eliciting a neuromuscular response from the vibrated muscles [72–76]. It has been shown that the vibration is dampened as it is transmitted throughout the body [72,77], and it has been hypothesised that this is a consequence of increased neuromuscular activity [78,79], in addition to the natural flexibility of the musculoskeletal structures. The mechanism by which WBV elicits a neuromuscular response is unclear, though two main theories are generally supported: the TVR, and the Muscle Tuning Theory. Regardless of the specific mechanism, it is likely that the stretch receptor plays a central role in the response. This is discussed in greater detail in Section 3.2.

The latencies of the monosynaptic reflexes of the lower limb mediated via the stretch receptor, previously described in Chapter 1, have thus been used as a benchmark to inform the range of frequencies most commonly employed by the commercially available WBV systems [71]. For example, the latencies of the Achilles and patellar tendon reflexes are 35.2 and 21.0ms respectively [80]. Similarly, the average H-reflex latency of the calf muscle group is 30.3ms [81]. Thus a full contraction-relaxation cycle mediated by the stretch receptor will occur up to maximum excitation frequency of 28Hz for the calf muscle group, and 48Hz for the quadriceps muscle group. These frequencies therefore represent the physiological limit of the stretch reflex. Training at or above these frequencies may lead to an improvement in muscle strength and power [71]. Training below these frequencies may preferentially target slow twitch (Type I) muscle fibers frequencies, be less likely to result in muscle fatigue, and promote improvements in muscle function [69]. The frequencies and amplitudes of vibration most commonly used are therefore 5 - 50Hz, and 0 - 5mm respectively. The focus of this literature review is on those studies describing the acute and chronic neuromuscular and physiological adaptations associated with WBV.

3.1.3 Vibration Transmission

A basic mathematical model was developed by Yue and Mester [82] describing the internal loads and the effects of internal masses during the transmission of vibration to the human body during WBV. Limitations of the model are: (i) that it is for an input vibration along one dimension only, and (ii) it is for a fixed limb position. Furthermore, the authors concluded that a lumped mass-spring system cannot be expected to accurately model the complexities of the human body in-vivo. However, the principal finding of the model is that as the input frequency from the vibrator and the resonant frequency of the limb move away from each other, the elastic properties of the limb begin to dominate, as dictated by the magnitude of neuromuscular contraction, and the vibration is thus largely limited to these structures. They further describe a flow of energy between the muscle/tendon units and WBV platform, with some dissipation of energy and thus damping due to inefficiencies in the body [82]. The model thus suggests that resonance of the lower limb, and therefore risk of damage, may be avoided by altering the resonant frequency of the lower limb during WBV. Further in-vivo work principally carried out by Wakeling *et al.* [79, 83] has gone some way to describing the natural frequency of the lower limb. They have thus postulated the Muscle Tuning Theory, which is a mechanism to alter the natural frequency of a limb during vibration, by changing muscle stiffness through altered neuromuscular activity and therefore protect against resonance. The Muscle Tuning Theory is described in greater detail in Section 3.2.2.

Vibration is a known occupation hazard and a greater understanding of lower limb natural frequencies is thus required to understand the potential for harmful effect from WBV. Some reported work has been carried out to this end [78, 79, 83, 84]. The damped natural frequency of the Triceps Suræ, Tibialis Anterior and Quadriceps were systematically quantified for a range of joint angles and levels of neuromuscular contraction. Average values were 16, 25 and 10Hz for each muscle respectively at a 0° joint angle, or 14, 26 and 10Hz at a 40° [83]. Each of these values easily fall within the input frequency range of typical commercial WBV platforms. However, during an isometric contraction these values increase to a value normally outside the WBV platform input range. Depending on the magnitude of the neuromuscular contraction, natural frequency values between 32 - 26Hz for the Triceps Suræ, 31 - 49Hz for the Tibialis Anterior, and 26 - 31Hz for the Quadriceps were found [83]. It would thus be naturally expected that increased tone or an instance of clonic spasm in an SCI participant would result in similar increments in muscle natural frequency.

Further work has thus been carried out to advise on the risks and benefits of WBV training and the dose that should be applied [85, 86]. A summary of the guidelines includes: (i) vibration to the head should be minimised by standing with a flexed knee; (ii) some resonance can occur below 20Hz, therefore exposure to frequencies below this value should be kept to a minimum; (iii) vibration amplitudes between 1 - 2mm should be applied as a starting point and only increased for training purposes, (iv) bouts of vibration should not exceed 60s; and (v) those with coronary heart disease or hypertension should be excluded [86].

3.2 Neural Mechanism

It is evident that WBV elicits an increase in neuromuscular activity from the Electromyogram (EMG) studies reviewed in later sections. Some have hypothesised that the increased muscle activity is related to postural control mechanisms, or descending vestibulospinal commands, that are responding to the mechanical disturbances caused by the vibrating platform [75,87]. However, the Tonic Vibration Reflex TVR, and the muscle tuning theory are more generally accepted, each of which are described in greater detail in Sections 3.2.1 and 3.2.2 respectively.

Regardless of the mediating mechanism, it is likely that the muscle spindle plays a central role in the detection and response to WBV. A novel experiment by Ritzmann *et al.* [88] showed that a standard blood pressure cuff placed around the soleus suppressed the short latency stretch reflex and the neuromuscular response to WBV. It was concluded that this suppression was most likely attributable to a decrease in muscle spindle sensitivity, rather than changes to the Ia afferent nerve, α -motoneuron or muscle fibre [89].

Vibration induced modification of motor unit excitability is discussed in Section 3.3.3. The excitability of a motor unit is most easily investigated in two ways: electrical stimulation of the common nerve or tendon percussion. These types of investigation provide an insight into the mechanism of muscle activation during WBV. The results presented in Section 3.5.1 indicate that pre-synaptic changes to the α -motoneuron are responsible for the acute improvements in muscle performance commonly found immediately after a bout of WBV.

The magnitude of the neuromuscular response to WBV is typically larger if the vibration is superimposed on a voluntary contraction or if the muscle is being stretched, suggesting that the response is moderated by muscle spindle sensitivity [75,85,90]. Spindle sensitivity is normally increased through increased fusimotor drive during voluntary contraction, or by muscle lengthening. Therefore, the potentiation of motor unit excitability post-WBV may also be a result of increased γ -motoneuron activity caused by the repetitive shortening and lengthening of the muscle during WBV.

3.2.1 Tonic Vibration Reflex

The application of high frequency, low amplitude vibration to skeletal muscle has been extensively investigated since the 1960s and is reasonably well understood. Vibration applied to a muscle results in an increase in α -motoneuron activity and a tonic contraction, thus called the tonic vibration reflex. It is most readily elicited by direct application of vibration to the muscle belly or tendon [91–98], though it has been shown that indirect application can also be used [91]. Vibration frequencies between 20 - 220Hz, and amplitudes between 0.5 - 3.3mm [94] are commonly used, though amplitudes up to 25mm have tentatively been reported [95]. A larger TVR is generally found with higher frequencies of vibration [94].

Primary endings (Type Ia) of the stretch receptor are particularly sensitive to vibration, whose excitation initiates a volley of Type Ia afferents [91–94, 97, 99]. Secondary spindle endings (Type II) and the Golgi Tendon Organ (Type Ib) are also excited by vibration, though to a much smaller extent (cf. Chapter 1) [91–93, 99]. Cutaneous receptors in the

skin are stimulated by vibration also, but have no motor output as a consequence, and are thought to play a role in the detection of vibration only [64].

Microneurography studies, which are in good general agreement, have found that muscle receptor afferents can be ‘driven’ by the frequency of vibration. Primary spindle endings exhibit better entrainment than secondary endings or the Golgi tendon organ [91, 93, 97]. Furthermore, different receptors were found to have a finite frequency to which they can be driven. Type Ia afferents have been driven to between 120 - 220Hz, compared to 40 - 50Hz for Type II and Ib afferents [64, 91, 93]. The preferential recruitment of primary spindle endings over the secondary endings and Golgi tendon organ has been shown at lower amplitudes of vibration [92]. In addition, the force with which the vibrator is applied strongly influences the TVR [93]. Vibration amplitude and application force must therefore be carefully considered when comparing the results of studies that used a hand-held vibration device. In summary, Type Ia afferents most likely mediate the TVR due to the larger sensitivity and better entrainment of the primary spindle endings to vibration.

Primary spindle endings fire once per vibration cycle in a relaxed muscle. There is a 1:1 relationship between the frequency of vibration and the firing frequency of the primary spindle ending up to the spindle frequency limit [91, 93]. The spindle is driven at a subharmonic of the frequency of vibration with a relationship of 2:1 when the spindle frequency limit is exceeded. The primary ending can fire multiple times during a single vibration cycle during muscle lengthening, so long as the spindle firing limit has not been exceeded [91].

The TVR is small in comparison to the maximum voluntary capacity of the muscle. It is characterised by its relatively slow onset and cessation, and gradual increase in magnitude to plateau within 20 - 60s of initiation [95]. This is fundamentally different to the ‘dead-beat’ ending of the myotatic or H-reflex, each of which exhibit a characteristic onset latency. The TVR is readily elicited in muscle that is relaxed, with better compliance during increased muscle stretch or if it is preceded by a tonic contraction [91, 94]. Good motor unit synchronisation up to a frequency of vibration of 100Hz has been shown [100] though an asynchronous motor unit firing frequency has been found also [95]. Finally, the TVR can be potentiated (for example by the Jendrassik manoeuvre) and voluntarily depressed [96]. In summary the factors which influence the TVR include [64]:

1. The location of vibration application [91].
2. The excitability of the central nervous system [96, 98].
3. The initial length of the homonymous muscle [94].
4. The vibration parameters (frequency and amplitude).

These characteristics indicate that the TVR is mediated via a polysynaptic pathway which uses some of the peripheral neural components involved in monosynaptic reflexes. The fact that the TVR can be voluntarily suppressed or potentiated indicates input from higher spinal segments [94].

Central nervous system influences has been shown by a number of studies. The TVR has been suppressed by barbiturates, which selectively inhibit interneuron activity, but with no effect on the myotatic or H-reflex. This therefore implies interneuron involvement during the TVR [95,96]. The TVR was found to be suppressed in human subjects with an ipsilateral cerebellar deficit when compared to the normal contralateral limb, therefore suggesting a cerebellar influence also [96]. It is noted however that cerebellar influence has not been shown in cats [64]. Posture has also been shown to alter the reflex response, suggesting a vestibulospinal effect [94]. Furthermore, the TVR has been shown to be considerably depressed in individuals with chronic SCI and those in spinal shock [98].

Vibration of the agonist inhibits α -motoneuron activity of the antagonist (reciprocal inhibition). Simultaneous vibration of the agonist and antagonist suppresses the TVR in both [64]. The myotatic reflex and H-reflex have both been shown to be depressed during vibration [95,96,98,101], which is unusual because the TVR, myotatic reflex and H-reflex are all thought to have some common neural pathways. This phenomenon has thus come to be described as the ‘vibration paradox’ [102], for which several causes have been proposed.

Spindle occlusion, the so called ‘busy line’ effect, was initially suggested as the cause of the vibration paradox [91]. It was proposed that the spindle output was inhibited and unable to respond to a tendon percussion or electrical impulse, having been so recently stimulated by vibration, particularly if it was firing at its maximum frequency. This theory has been refuted by others who suggests that such occlusion would be insufficient to fully suppress the myotatic or H-reflex [64]. Assuming that the same motoneuron pool is used for each of these reflexes, it was shown that vibration does not affect the post-synaptic pathway, and hypothesised that pre-synaptic inhibition due to repetitive stimulation to the Ia afferent is the dominant factor in suppressing the monosynaptic reflexes during vibration [99]. Pre-synaptic inhibition may be due to altered muscle spindle sensitivity [97], however despite this the authors concluded that central nervous adaptations are more likely to be the principal mediating factor.

Pre-synaptic inhibition alone does not explain how a tonic contraction can be maintained by vibration, yet inhibit reflexes that use the same neural networks. Desmedt and Godaux [102] proposed that the TVR recruits individual motor units according to the Henneman size principle and that pre-synaptic inhibition acts in reverse order on the same motor units. This would thus limit the total number of motor units that could potentially be recruited by the monosynaptic reflex. In addition, this may explain the typically small magnitude of the TVR.

In contrast, the myotatic reflex is potentiated after vibration, whereas the H-reflex continues to be depressed [96,97,101,103]. Post-tetanic potentiation of the myotatic reflex may be occurring after vibration, whereby Ia afferents are being selectively recruited by the tendon percussion [103]. On the other hand, electrical stimulation of the mixed nerve to elicit a H-reflex will preferentially recruit Ib over Ia afferents, which will result in an inhibited motor response. The authors proposed that this is because the firing threshold of the primary spindle endings increased due to the repetitive stimulation of the vibration. Meanwhile, the resting membrane potential of the Golgi tendon organ remained unchanged, due to its relative

insensitivity to vibration. The Ib afferents were thus preferentially recruited by the electrical stimulus applied after a period of vibration [103].

There are a number of similarities between the neuromuscular response to the direct application of vibration and the neuromuscular response to WBV. Each has an asynchronous motor response, and each similarly moderates the short latency reflexes (cf. Section 3.3.3). However, the frequencies and amplitudes of vibration are considerably different. In addition, the transmission of vibration throughout the body during WBV is poorly understood. It is not known if the vibrations that are ultimately detected by the muscle during WBV are comparable to those detected during the direct application of vibration. An alternative theory has therefore been proposed. The muscle tuning theory is described in the following section [104, 105].

3.2.2 Muscle Tuning Theory

The muscle tuning theory was first proposed as a mechanism which moderates neuromuscular activity during walking with the function of dampening the transmission of vibration to the body elicited at heel strike during walking and running [106]. Heel strike has a frequency and amplitude component, and is thus analogous to the mechanical displacement experienced during WBV. The frequency component of heel strike falls in the range of 10 - 20Hz and it was shown that the soft tissue of the lower limb could potentially resonate within this frequency range [83]. The natural frequency of a given muscle was found to be between 8 - 33Hz depending on joint angle. Furthermore, the natural frequency increased to between 19 - 59Hz when the homonymous muscle was under contraction. The ‘muscle tuning’ theory was thus proposed as a method of moderating the natural frequency of muscle, dampening the transmission of vibration, and minimising the risk of resonance and damage to the musculoskeletal structures [78].

A ‘feed-forward’ mechanism was proposed in a later study, whereby a muscle contraction was initiated before heel strike. This is advantageous for two reasons: the musculoskeletal system is sufficiently stiff and able to support the body during stance, and sufficient time has passed to generate adequate tension in the muscle to alter its natural frequency [79]. More recently, it has been suggested that the muscle tuning response is mediated via the fast twitch muscle fibres, which can more rapidly act to change muscle tension [84]. This theory is supported by others who found the largest neuromuscular response of the rectus femoris occurred at a vibration frequency of 23Hz on a WBV platform [107]. This same frequency was shown to be the resonant frequency of the muscle.

Notwithstanding the lack of agreement with regards to the specific neural mechanism by which WBV elicits an increased neuromuscular response from muscle, and the relative infancy of the muscle tuning theory compared to the TVR, a period of WBV does elicit a number of acute (Section 3.5) and chronic (Section 3.6) physiological changes. These changes have been investigated for neurologically intact and neurologically disabled populations.

3.3 Acute Neuromuscular Response

Investigation into the acute neuromuscular response to WBV has predominantly been carried out within the last ten years. These studies have focussed on the lower limb and the back, where the neuromuscular response of the tibialis anterior, gastrocnemius, soleus, rectus femoris, vasti, biceps femoris, gluteus, and spinae erector muscles have been investigated. Methodological differences in protocol, data recording and off-line analysis makes between-study comparisons difficult. In terms of protocol, differences include: (i) the type of vibration platform used (SA-WBV, VV-WBV), (ii) exercise type (static, dynamic), and (iii) test population (general, athletic, elderly, neurologically-compromised).

In terms of data recording and off-analysis, differences include: (i) the type of EMG electrode used (active, passive), (ii) data normalisation with respect to a maximum voluntary contraction, and (iii) off-line processing of the EMG (cf. Section 3.4). Consideration of these differences in protocol is particularly important when interpreting EMG recorded during WBV. It is now generally accepted that these EMG contain movement artefact, and if neglected will lead to an overestimation of the magnitude of neuromuscular activity during WBV. Abercromby *et al.* [75] were the first to advocate the targeted removal of artefact, thereby calling into question the interpretation of the results from previous EMG studies. Notwithstanding these differences however some general conclusions can be drawn.

WBV typically elicits an increased neuromuscular response. Larger increases are generally found in those muscles that are located closer to the vibrating platform [72,75,108], muscles that are under contraction [109], or muscles that are stretched [110]. For example, the increased activity induced in the gastrocnemius is normally larger than that induced in the gluteus [75]. Furthermore, a unilateral static squat will elicit a larger neuromuscular response than a bilateral squat during WBV [90]. Neuromuscular activity normally increases with an increase in vibration amplitude [72,73], or frequency [72], with maximal responses reported to occur between 23 - 45Hz [76,107,109].

This section begins by describing the acute neuromuscular response of those in the general population to a short bout of WBV. Those studies with a protocol requiring the support of additional load during WBV are described separately to those who applied WBV in a more conventional manner. The response of those with a SCI are discussed next. Studies that investigated the effect of WBV on the short latency reflex are described at the end.

3.3.1 General Population

Vibration Only

Fatigue has been implicated by SA- and VV-WBV [111,112], whereby a decrease in the mean power frequency of the EMG was found as the bout of WBV progressed. It is noted however that the possible presence of movement artefact or motor-unit entrainment were unaccounted for in this analysis, each of which could have occurred and potentially result in a decrease in mean power frequency, and would therefore not necessarily be an indication of fatigue. In a similar study, the maximal neuromuscular response of the vastus lateralis during unloaded, static squats occurred at the 30Hz and 5mm vibration on a VV-WBV device [76].

The neuromuscular response of the quadriceps and calf muscle groups were measured during unloaded, unilateral- and bilateral- static squats while vibration was applied by a similar platform in a later study by Roelants *et al.* [90]. A similar frequency ($f = 35\text{Hz}$), but smaller amplitude ($A = 2.5\text{mm}$) of vibration was applied. A contraction up to 83% Maximum Voluntary Contraction (MVC) was found during WBV when in a deep- or unilateral-squat, each of which were found to result in a considerably larger response than a bilateral shallow squat. Marin [73] found that the magnitude of the neuromuscular response of the quadriceps and calf muscle groups increased with vibration amplitude, whilst performing static, unloaded squats on a VV-WBV platform. Footwear did not affect the neuromuscular response of the quadriceps, but augmented the response of the calf. Passive EMG electrodes were used in each of these studies. The presence of vibration induced artefact in the EMG was not accounted for in the respective analyses and an overestimation of the neuromuscular response to WBV is likely (cf. Section 3.4).

Abercromby *et al.* [75] found a larger neuromuscular response from the vastus lateralis, biceps femoris and gastrocnemius during SA- versus VV-WBV, at a frequency and amplitude of 30Hz and 4mm respectively. They found that static squats elicited a larger response from the vastus lateralis and gastrocnemius than dynamic squats, supporting other findings that a voluntary contraction augments the neuromuscular response to WBV. They suggested that the human body responds to WBV via a postural control mechanism, and hypothesised that the difference in response between the two devices is related to the fact that the principal component of vibration during VV-WBV is in the z-direction only, whereas it is in the y- and z-directions during SA-WBV (cf. Figure 3.2). Surprisingly, they found that a shallow squat elicited a larger response than a deeper one. This is in contrast to findings from others who found no difference or a slightly larger neuromuscular response with increasing knee flexion [90, 110].

A positive, linear relationship between the neuromuscular activity of the shank muscles and the frequency of SA-WBV was identified by Pollock *et al.* [72]. A small increase, that was quick to plateau was found for the rectus femoris and biceps femoris. The activity of the gluteus was found to increase with vibration, but no difference in response was identified for different vibration frequencies. This data is supported by acceleration measurements which showed a reduction in the transmission of vibration with increasing proximity to the WBV platform. It was concluded that both frequency and amplitude of vibration moderate the neuromuscular response, and that the lower limb considerably dampens the transmission of vibration to the upper body.

Vibration with Additional Load

An exhaustive bout of SA-WBV, during which the participants performed dynamic squats while carrying an additional load (40% body-weight) for 4 minutes, was found to decrease the mean frequency (Equation 3.2) as recorded from the vastus lateralis during a post-vibration maximum voluntary contraction [113].

$$MNF = \frac{\sum_{i=1}^{f_N} f_i P_i}{\sum_{i=1}^{f_N} P_i} \quad (3.2)$$

Where MNF = Mean frequency; N = Number of Frequencies; f_N = Maximum Frequency; f = Frequency; P = Power; i = i th line in power spectrum analysis.

This suggests that resistive vibration exercise has a fatiguing effect on muscle. More recently, Hazell *et al.* [109] found that an additional load of 30% body-weight during VV-WBV increased the acute neuromuscular response by 2.5 - 3.5%. Marin *et al.* [114] found that increasing frequency during unloaded, static squats on a VV-WBV platform was equivalent to increasing the load carried during static squats without WBV. They concluded that platform acceleration moderates the neuromuscular response to WBV, and that a platform acceleration of 1.2 - 10g is equivalent to an increased load of 20 - 70kg during a static squat.

3.3.2 Spinal Cord Injured Population

Relatively few studies to date have investigated the acute neuromuscular response of SCI patients to WBV. Herrero *et al.* [115] found that SA-WBV implemented with a tilt-table elicited an increased response from the vastus lateralis and vastus medialis of motor-complete SCI (CSCI) patients (AIS A), but no difference between the frequencies was found. Chang *et al.* [87] found no change in the neuromuscular activity of chronic CSCI patients (AIS A) to VV-WBV while seated. They suggested that descending input from the vestibular system may be moderating the neuromuscular response to WBV, and that the lack of excitation of this system in a seated position accounts for the absence of any response.

3.3.3 Short Latency Reflex

The short latency reflex (cf. Chapter 1) is a monosynaptic reflex that is most readily elicited via a tendon percussion (myotatic reflex) or electrical stimulation of a spinal root (H-reflex). It is measured as an EMG and is characterized by its onset latency and magnitude. In the lower limb, the myotatic reflex is most readily elicited in the hamstring via an anterior-posterior tibial translation, in the quadriceps via tendon percussion of the patellar tendon, or in the calf muscle groups via tendon percussion of the Achilles tendon. The H-reflex can be most readily elicited in the calf muscle group by electrically stimulating the tibial nerve via a transcutaneous electrical stimulation electrode placed at the popliteal fossa. The short latency reflex provides an indication of the excitability of the motoneuronal pool supplying the homonymous muscle group. It has thus been used as an outcome measure from numerous studies investigating the acute neuromuscular response to the direct and indirect application of vibration. This section describes the effect of an acute bout WBV on the short latency reflex.

Significant decreases in the H-reflex, H_{max} and the H_{max}/M_{max} ratio, have been reported in the soleus of able-bodied and SCI subjects post-WBV [116–119]. Armstrong *et al.* [120] monitored the rate of recovery of the H-reflex in able-bodied subjects, and found it to be completely suppressed in the first minute post-WBV. Thereafter, they classified recovery into four groups: (i) the first group exhibited full recovery of the H-reflex back to baseline within 3mins post-vibration; (ii) the second group recovered within 7mins; (iii) the third group within 15mins; (iv) and the fourth group showed no recovery within 30mins post-WBV. Beekhuizen *et al.* [116] initially showed that standing on a SA-WBV platform for 5

minutes significantly reduced the H-reflex amplitude by 25%. Kipp *et al.* [118] later showed that the rate of recovery of the amplitude of the H-reflex was between 1 - 2min, though a VV-WBV platform was used in this later study. This was corroborated by a further study carried out by Sayenko *et al.* [117], however the difference in the rates of recovery from the two different studies likely stems from the fact that the initial study applied vibration at 25Hz and 2 - 4mm for 5 minutes, compared to 35Hz and 1mm for 1min. Furthermore, Sayenko *et al.* [117] recorded the rate of recovery by measuring the H-reflex every 6s. Recommendations are that no more than one H-reflex be elicited for every 10s period [55]. By contrast Kipp *et al.* [118] measured the H-reflex every 15s thus lending greater confidence in their findings. Chang *et al.* [119] demonstrated an 83% suppression of the H-reflex which lasted in excess of 2mins. In this study, the vibration was applied from a seated position, rather than standing as was the case in each of the other studies. Furthermore, the H-reflex was measured both during and after the application of vibration. There was a high likelihood of sensor movement during vibration, and thus a comparison of H-reflex depression between vibration and when it has stopped is not valid because it cannot be guaranteed that the measurements were taken from the same motor-units. In addition to the methodological differences highlighted here, Armstrong *et al.* [120] suggest that differences in muscle fiber composition due to age, gender, training specificity and conditioning status may each contribute to the variable rates of recovery observed in these studies.

Conversely, no change in α -motoneuron excitability was found in a similar study by McBride *et al.* [121]. However, an increase in the maximum force generating capacity of the triceps surae was identified after an acute bout of WBV. Rittweger *et al.* [122] found that the stretch reflex was potentiated post-WBV, but Hopkins *et al.* [123] did not. In a well controlled experiment they found no change in electromechanical delay or the latency of the stretch reflex either [123].

The H-reflex was inhibited in SCI subjects and completely suppressed in able-bodied subjects during a bout of WBV [117]. Notwithstanding the methodological flaws (cf. Section 3.4), that failed to take into account the possibility that the detection electrodes may have moved during vibration, thus perhaps attributing to the reduced H-reflex, the acute changes in the reflex amplitude during WBV do bear a resemblance to those changes that occur during the TVR.

This confounding body of evidence has led some to suggest that reduced post-WBV muscle performance is due to pre-synaptic inhibition of the primary ending of the muscle spindle [120], or neurotransmitter depletion [117]. Others suggest that this is not the case and post-tetanic potentiation, a build up of calcium at the axon terminal of the presynaptic neuron [121], or altered proprioception leading to enhanced postural control [123] is occurring. Reduced short latency reflex excitability does seem to contradict the post-WBV potentiation of muscle performance. Kipp *et al.* [118] have proposed that there is a reduction in homosynaptic depression post-WBV, which is a rate-dependant response that moderates the excitability of the Ia afferent- α -motoneuron synapse.

3.4 Vibration Artefact

Vibration artefact is manifested as a series of sharp ‘spikes’ in the power of the EMG when it is viewed in the frequency domain. These spikes in power occur at the frequency of vibration and to a lesser extent at its harmonics and are induced in the EMG by movement during vibration. Failure to take account of vibration artefact could result in an overestimation of neuromuscular activity and impact upon the interpretation of any EMG results taken during WBV, such as those studies described in Section 3.3. This section presents the research which has investigated this form of vibration artefact.

The presence of movement artefact in an EMG recorded during WBV is indicated by an atypical Power Spectral Density (PSD) plot or amplitude spectral density plot. Figure 3.3 outlines two sample EMG presented in the frequency domain. The first was recorded prior to WBV, and the second was recorded during WBV ($f = 20\text{Hz}$). Clear increases in the power of the EMGs recorded during WBV can be seen across all frequencies, with distinct spikes in signal power at harmonics of the frequency of vibration. In some instances, these harmonic frequencies have been found to increase the power of the EMG by 300% [72]. Abercromby *et al.* [75] were the first to suggest that these distinct spikes in signal power were vibration induced movement artefacts and advocated their removal. All EMG studies prior to this did not address the possible presence of movement artefact [73, 76, 88, 90, 111, 117, 124], and most investigators are now in general agreement that they should be removed [72, 107–110, 114, 115, 125]. Sharp, stop-band filters are most commonly used with a stop-band width of 1.5 - 3Hz [75, 107, 126]. Typically the first three harmonics of vibration are removed [107, 108, 126], though in some cases up to fifteen have been removed [75, 114, 115]. Others have implemented dual-pass butterworth filters [109], or spectral smoothing [125].

The magnitude of the artefact induced in an EMG recorded from the quadriceps during VV-WBV was quantified by Fratini *et al.* [107, 126]. An EMG electrode was placed on the comparatively electrically inert patella in addition to EMG recordings from the vastus medialis, vastus lateralis and rectus femoris. Acceleration was also measured from the skin over the rectus femoris. Frequency analysis identified that the frequency of vibration, as recorded from the rectus femoris, corresponded exactly with the artefact signal in the EMG. The signal-to-noise ratio was estimated by comparing the signal as recorded from the patella, to that recorded from the muscle. Artefacts were found to constitute up to 30% of the signal power, and their magnitude typically increased with frequency.

These spikes in the EMG have been described to as stretch reflexes rather than motion artefact by Ritzmann *et al.* [88]. It was hypothesised that ‘phase-locking’ was occurring. During phase-locking the muscle activity is synchronised with the frequency of the vibrating, resulting in an increase in signal power specifically at the frequency of vibration. Indeed this view is supported by evidence from the tendon vibration studies by Martin and Park [100] who showed good motor-unit entrainment up to 100Hz during the direct application of vibration to the tendons of the hand flexor muscles.

It was thus proposed that the experiments by Fratini *et al.* [107, 126] were flawed because

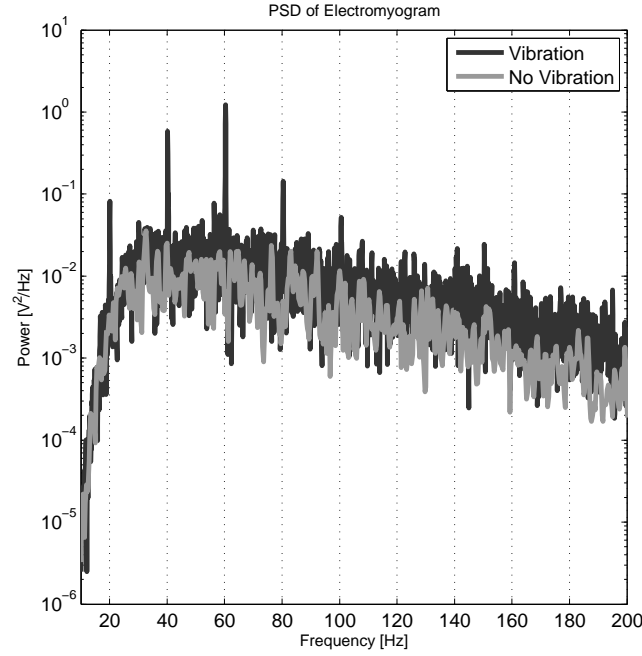


Figure 3.3: Example of power spectral density plots for electromyogram data that was recorded without WBV (No Vibration), and during WBV (Vibration). The power spectral density plots are derived from raw data acquired during the study presented in Chapter 5. This raw data was recorded at the 20Hz vibration condition. PSD: Power spectral density.

an EMG recorded from the patella would likely detect cross-talk from the adjacent muscle groups [88]. Furthermore Ritzmann *et al.* [88] identified a neuromuscular response during WBV which had a latency that was in good agreement with that of the stretch reflex. They therefore concluded that filtering an EMG to remove ‘artefact’ was incorrect because it would inadvertently eliminate that neuromuscular activity which was phase-locked with the vibration. An alternative experimental setup using ‘dummy’ EMG electrodes was used instead. The dummy electrodes were electrically connected using a fixed resistor and then isolated from the skin. These electrodes were placed in close proximity to the recording-electrodes and were thus expected to receive the same dose of vibration as genuine EMG electrodes. It was hypothesised that the dummy electrodes would not detect the neuromuscular activity of the underlying muscle and would acquire movement artefacts only. This setup however, would detect only those voltage potentials generated through the movement of the EMG cable, and would neglect other potential sources, which include: (i) the relative movement of the bi-polar electrodes with respect to each other, (ii) the relative movement of the electrodes with respect to the skin, (iii) the relative movement of the skin with respect to the muscle, and (iv) spatial impedance differences of the underlying tissue.

It is concluded from the evidence presented above that the frequency of vibration and its harmonics should be filtered to avoid an overestimation of the magnitude of the EMG. Furthermore, it is conceded that this approach is conservative and will result in an underestimation of the signal amplitude due to the removal of those frequency components that coincidentally occur at the frequency of vibration and its superior harmonics.

3.5 Acute Physiological Response

In addition to changes in the acute neuromuscular response during WBV, and some reported adaptation of the myotatic and H-reflexes, a number of other acute physiological changes in muscle performance have been investigated in the general population. These changes are described in this section. The available literature on similar adaptations in those with a SCI is particularly limited. A summary of the reported literature for this population is described below, in addition to describing the acute response of those with other forms of neurological deficit.

3.5.1 General Population

The acute effect of WBV on muscle performance remains equivocal. Changes in muscle power, force, and torque have been described, in addition to changes in jump height, grip strength, balance, flexibility, and stability. These studies have been carried out across a range of WBV platforms (VV-WBV, SA-WBV), exercise positions (static-squat, dynamic-squat), and loads. Typically frequencies between 20 - 40Hz, and amplitudes between 1 - 5mm have been applied.

Average force, power and velocity during an isometric squat and dynamic leg press have been found to remain the same or increase after an acute bout of WBV [127,128]. Cardinale and Lim [129] found no change in counter-movement jump height after an acute bout of VV-WBV at 20Hz, and a decrease in the same parameter after vibration at 40Hz. Cormie *et al.* [128] found an increase in counter-movement jump height after an acute bout of VV-WBV at 30Hz. A later study by Bazett-Jones *et al.* [130] could only show such increases in female participants, with no improvements found for the male participants. An exhaustive bout of SA-WBV with an additional load of 40% body-weight led to a decrease in squat jump height [113]. However, others have shown no change [112,129], or an increase [111,129] in squat jump height if the vibration exercise is performed without an additional load. Cochrane and Stannard [131] argue that squat jumps and counter-movement jumps are not normal during every day movement, and suggest that an arm counter-movement jump is a more representative test. They found that an acute bout of SA-WBV resulted in an increase in arm counter-movement jump height. Furthermore, they found that bicycling or a non-vibration condition resulted in a decrease in arm counter-movement jump height.

A follow up study that compared exhaustive SA-WBV with an additional load to a sham condition [122]. Similar exercises were performed during the sham condition but no vibration was applied. A similar decrement in squat jump height was found for both the vibration and sham conditions, but potentiation of the stretch reflex was found in those subjects that received the vibration treatment only. This finding was corroborated by others who identified a similar degree of potentiation of the short latency reflex of the hamstring after an acute bout of VV-WBV [132]. The metabolic demand of exhaustive SA-WBV was found to be moderate, where peak oxygen uptake values of 50% maximum oxygen uptake and an average heart rate of 130 min^{-1} were reported [113]. These values were smaller than those achieved during leg cycle ergometry. EMG analysis indicated that neuromuscular fatigue was occurring. Knee extension torque typically remained the same [112], or decreased [133] after an acute bout of WBV, particularly if the vibration session was performed with additional load [113,122].

Other findings showed that an acute bout of SA-WBV increased leg muscle stiffness and knee stability [132, 134]. However, others did not identify the same results for ankle stability [135]. Improvements in hamstring flexibility, stability and balance have also been shown [76, 111, 112, 129, 132].

Notwithstanding the wide and varied range of these studies and results, some tentative observations are made. Some aspects of muscle performance are potentiated after an acute bout of WBV: (i) most studies have found an increase in explosive muscle power as identified by an increase in jump height and squat power, (ii) the force producing capacity of the muscle normally decreases, and (iii) the stretch reflex is normally potentiated. The low metabolic demand and heart rate experienced during WBV exercise, has led to the conclusion that the fatigue manifested during WBV is neural in origin rather than muscular, and that WBV results in some potentiation of neural pathways. Furthermore, there exists a balance between neural potentiation and overall fatigue.

3.5.2 Neurologically Compromised Population

Studies describing the effect of an acute bout of WBV in those with a neurological deficit is limited. A summary of the literature summarised in this section is provided in Table 3.1. An acute bout of WBV has been shown to increase isometric and eccentric muscle torque in acute stroke patients [136]. The authors attributed the improvements to an increase in the rate of torque development, leading to a faster recruitment of the large motor units, and the relaxation of the antagonistic muscle, suggesting an acute reduction in tone [136]. Mean and peak blood flow were also found to increase by 11.3 - 23.0%, and 22.0 - 36.0% respectively, with increasing angles of tilt when WBV was applied on a tilt-table [115]. These increases in blood flow and neuromuscular activity were also associated with changes in gene expressions that are associated with neural plasticity [119]. The transmission of vibration to the head on a tilt table was 24 - 59% in able bodied controls, and 17 - 42% in those with an SCI [137]. It was also shown that the proportion of body weight carried by the lower limb when in this setup was 22 - 46%. A later study by Bernhardt *et al.* [138] showed that by using a standing frame, rather than a tilt-table, the weight carried by the lower limb increased to 76 - 85%. Each of these studies concluded that this evidence is sufficient to warrant further investigation of the musculoskeletal benefits of WBV training, either with the aid of a tilt-table or a standing frame [137, 138].

Table 3.1: Acute changes associated with whole body vibration in populations with a neurological deficit. Deficit duration: Time since stroke or spinal cord injury; VV: Vertically vibrating; SA: Side alternating; WBV: Whole body vibration; f: Frequency; A: Amplitude; a: a_{RMS} ; ♂: Male; ♀: Female; Age: Mean age of test population; ISO: Isometric; ECC: Eccentric; CTRL: Control; EMG: Electromyography; SCI: Spinal cord injury; Dose: Total duration of application of vibration; †: Statistically significant; ‡: Numerically estimated.

Author	Vibration param.	Intervention	Test Population	Test	Results
Tihanyi <i>et al.</i> [136]	VV-WBV Dose: 6mins f: 20Hz A: 2.5mm a: 2.8g‡	Seated. Static squats and standing	Stroke ♂♀ Age: 58.2yrs Deficit duration: 3.9wks 8 X WBV 8 X CTRL	a) ISO & ECC strength b) EMG	ISO: WBV (36.6%†); CTRL (8.4%). Rate of torque development: WBV (19%†); CTRL (10.9%). ECC: WBV (22.2%†); CTRL (5.3%). EMG: 44.9%†increase in vastus lateralis and no change in biceps femoris during ISO contractions. 33.2%†in vastus lateralis, and 22.5%†decrease in biceps femoris during ECC contraction. 13.1%†increase in median frequency.
Herrero <i>et al.</i> [115]	SA-WBV Dose: 3mins f: 10 - 30Hz A: 2.5mm a: 0.7 - 6.4g‡	Static squat on tilt table	SCI ♂♀ Age: 36.1yrs Deficit duration: 8.6yrs 8 X SCI	a) Blood flow	Peak blood volume increased 11.3 - 23.0%†. NS difference between the difference frequencies tested. Mean blood flow increased 22.0 - 36.0%†. Flow at 30Hz was found to be significantly larger than flow at 10Hz. Overall, blood flow increased with frequency.
Asselin <i>et al.</i> [137]	VV-WBV Dose: 4.5mins f: 34Hz A: 0.05 - 0.09mm‡ a: 0.19 - 0.31g	Standing on tilt table	SCI ♂ Age: 40yrs Deficit duration: 15yrs 7 X SCI 10 X CTRL (No neurological deficit)	a) Vibration transmission	Increased transmission of 24 - 59% in CTRL group, with tilt angle and platform acceleration. 17 - 42% transmission in SCI, with NS difference in transmission when comparing each group. 22 - 46% body weight borne through lower limb between 15 - 45 degrees of tilt.
Bernhardt <i>et al.</i> [138]	VV-WBV Dose: 5mins f: 34Hz A: 0.06mm‡ a: 0.3g	Standing with assistance of standing frame	SCI ♂♀ Age: 40yrs Deficit duration: 11yrs 11 X SCI	a) Ground reaction force	Ground reaction force of 76 - 85% in motor complete spinal cord injured participants during WBV in a standing frame.

3.6 Chronic Physiological Adaptations

This section reviews the chronic physiological adaptations of muscle that have been associated with the period of extended WBV training. The section begins by reviewing the response of the general population, and concludes by reviewing the response of sample populations with a number of differing types of neurological deficit.

3.6.1 General Population

The application of WBV as a training modality has been described for a broad range of populations with no neurological deficit. Typical test subjects include: (i) non-athletic, young men and women (< 35years) [124,139–149]; (ii) moderately-trained and sprint-trained, young men and women (< 35years) [150,151]; (iii) non-athletic, elderly women (> 65years) [152–157]; and (iv) athletic and non-athletic, elderly men (>65years) [158,159].

Muscle strength and power are the main outcome measures typically used to describe the effect of WBV training, and are most commonly quantified by: (i) isometric knee flexion and extension flexion torque [67, 124, 139, 140, 146, 147, 151, 153, 157, 158, 160]; (ii) isokinetic knee flexion and extension torque [140, 143, 145, 146, 148, 151, 153, 160]; (iii) maximum voluntary contraction [69,141,144,149,161]; (iv) maximum electrically elicited force generating capacity [141, 145]; (v) repetition maximum [144, 148, 150, 155]; (vi) counter movement jump height [124, 139–142, 144, 145, 147–153, 157]; (vii) squat jump height [142, 145, 149]; and (viii) multiple vertical jumps [149]. Other outcome measures include: (i) sprint speed/shuttle run [124, 139, 142, 149, 151]; (ii) body fat/body mass index/fat free mass [143, 146, 148, 160]; (iii) muscle cross sectional area [67, 155, 157, 161]; (iv) fiber phenotype [69, 147]; (v) balance/postural sway [124,139,154,159]; (vi) agility [142]; (vii) grip strength [124,139]; (viii) bone mineral density [124,152]; (ix) timed up-and-go [155]; (x) maximal cycling power [147]; and (xi) respiratory parameters [147].

The frequencies and amplitudes of vibration typically used are between 25 - 50Hz and 1 - 5mm respectively. Some studies have investigated frequencies below 8Hz [149,158], and amplitudes as low 0.1mm [152], and in some instances, these parameters are used as sham vibration conditions. Note that the same exercises are carried out during a ‘sham vibration’ condition but without WBV, or WBV at very low intensities. Platform accelerations during sham vibration are therefore between 0 - 0.05g [158], however the majority of studies describe platform accelerations between 1.7 - 18g. Training programmes are typically carried out over 2 - 32 weeks, though some have been carried in excess of 52 weeks [157,159,160]. Training is normally carried out 3 - 5 times per week, though a small number of studies have carried out training in excess of 10 times per week [67,69]. The total dose of vibration therapy per training session is typically reported to be between 2 - 30 minutes, with the optimum dose suggested to be approximately 15 minutes [162]. As endurance and tolerance builds over the course of the training programme, overload is typically achieved by increasing the frequency or amplitude of vibration, or by increasing the total dose of vibration during a session.

The majority of studies describe training programmes during which the participants perform

a series of standing exercises, jumping exercises, static or dynamic squatting exercises, heel-lifts, repetition maximum, or a combination of these exercises, in a standing position on a WBV platform. These exercises can be carried out with, or without additional load. Other studies have investigated the potential of WBV training to counteract musculoskeletal decline during prolonged bed-rest [67, 69, 161], and the application of WBV training from a seated position [144].

Some studies, particularly those first published, used a control group that did not perform any training for comparison with those who did [67, 124, 139, 147, 148, 152, 154, 155]. This led to criticism of some of the early work as it is difficult to determine the efficacy of WBV training, or to ascertain to what extent any positive effect reported is attributable to WBV and to the training intervention alone [163]. More recent investigations have used sham-vibration groups as controls, whereby the same intervention is carried out but in the absence of vibration [140–142, 144, 146, 148, 150, 160, 161]. Others have compared a WBV training programme to conventional fitness or strength training programmes [140, 143, 151, 153, 159]. A small number of studies have compared the training effect of different frequencies of vibration [145, 149, 158]. The original research articles reviewed in this section are summarised in Table 3.2 - 3.4. These tables outline the vibration parameters, training parameters, test population, control groups, outcome measures, and significant results. The results are discussed in greater in the next section.

Table 3.2: Chronic changes associated with whole body vibration in general populations with no neurological deficit

Author	Vibration param.	Training param.	Intervention	Test Population	Test	Results
Torvinen <i>et al.</i> [139]	VV-WBV f: 25 - 40Hz A: 1mm a: 1.7 - 4.5g	16 weeks 3 - 5 sessions/week 4 mins/session	Multiple static and dynamic exercises	Non-athletic ♂♀ Age: 29yrs 26 X No intervention 26 X WBV	a) CMJ b) ISO knee strength c) Grip strength d) Shuttle run e) Postural sway	CMJ: WBV (10%†). ISO knee extension strength: WBV (3.7%†). NS changes found for all other tests.
Russo <i>et al.</i> [152]	SA-WBV f: 28Hz A: 0.1mm† a: 0.3g	24 weeks 2 sessions/week 6 mins/session	Static and dynamic squats	Non-athletic ♀ Age: 61yrs 15 X No intervention 14 X WBV	a) CMJ b) BMD	Power: WBV (4.7%); No Intervention (-0.4%). Muscle force: WBV (0.1%); No intervention (2.6%). Force velocity: WBV (4.9); No intervention (-1.5%). Significant difference in velocity and power between groups. NS differences in BMD.
Delecluse <i>et al.</i> [140]	VV - WBV f: 28Hz A: 1.2 - 2.5mm a: 2.2 - 5g	12 weeks 3 sessions/week 3 - 20 mins/session	Static and dynamic squats	Non-athletic ♀ Age: 22yrs 20 X WBV 21 X Sham-WBV 20 X RES training* 13 X No intervention	a) ISO knee strength b) CMJ	ISO knee extension strength: WBV (13.6%†); RES (14.4%†). DYN knee extension strength: WBV (9%†); RES (7%†). NS difference in strength post-WBV or RES. CMJ: WBV (7.6%†). Sham-WBV and no intervention showed no training benefit.
Torvinen <i>et al.</i> [124]	VV-WBV f: 25 - 40Hz A: 1mm a: 1.7 - 5.7g	32 weeks 3 - 5 sessions/week 4 mins/session	Static and dynamic squats	Non-athletic ♂♀ Age: 29yrs 27 X WBV 26 X No intervention	a) CMJ b) BMD c) ISO knee strength c) Grip strength d) Shuttle run e) Postural sway	CMJ: WBV (7.8%†). No change in BMD, biochemical markers ISO knee extension strength, grip strength, shuttle run, or postural sway.
deRuiter <i>et al.</i> [141]	SA-WBV f: 30Hz A: 4mm a: 10g†	11 weeks 3 sessions/week 5 - 8 mins/session	Static squats	Non-athletic ♂ Age: 20yrs 10 X WBV 10 X Intervention only	a) CMJ b) MFGC c) MVC	No significant increase in bi-lateral knee extension strength, MFGC, or CMJ found in either group. Larger tendency for rate of force rise identified in WBV group.
Roelants <i>et al.</i> [153]	VV-WBV f: 35 - 40Hz A: 1.25 - 2.5g a: 2.2 - 5.09g	24 weeks 3 sessions/week 3 - 30 mins / session	Static and dynamic squats	Non-athletic ♀ Age: 65yrs 24 X WBV 20 X RES training* 25 X No intervention	a) CMJ b) ISO and DYN knee strength	ISO knee extension strength: WBV (12%†); RES (16%†). DYN knee extension strength: WBV (12%†); RES (12%†). CMJ: WBV (12% †); RES (16%†). NS difference between groups and no intervention had no training effect.
Cochrane <i>et al.</i> [142]	SA-WBV f: 26Hz A: 5.5mm a: 10g†	2 weeks 4 - 5 sessions/week 10 mins/session	Static squats	Non-athletic ♂♀ Age: 24yrs 12 X WBV 12 X Intervention only	a) CMJ b) SJ c) Agility d) Sprint	JH: WBV (4.3%). CMJ: WBV (3.9%). NS difference between groups and decrease found in intervention only group. No change in agility or sprint speed in either group.
Roelants <i>et al.</i> [143]	VV-WBV f: 35 - 45Hz A: 1.25 - 2.5mm a: 2.2 - 5g	24 weeks 3 sessions/week 3 - 20 mins/session	Static and dynamic squats	Non-athletic ♀ Age: 21yrs 13 X WBV 15 X RES training* 12 X No intervention	a) DYN knee strength b) FFM	DYN knee extension strength: WBV (5.9 - 24.4%†); RES (10.2 - 16.5%†). NS difference between groups. FFM: WBV (2.2%†). No change in body weight or fat in any group.
Rønnestad [150]	VV-WBV f: 40Hz A: NR a: NR	5 weeks 2 - 3 sessions/week NR	90 - 100% RM	Moderately-trained ♂ Age: 31yrs 8 X WBV 8 X RM only	a) CMJ b) RM	RM: WBV (32.4%†); RM (24.2%†). NS difference between groups. CMJ: WBV (9.1%†) RM (4.2%).

Table 3.3: Chronic changes associated with whole body vibration in general population with no neurological deficit (continued)

Author	Vibration param.	Training param.	Intervention	Test Population	Test	Results
Delecluse <i>et al.</i> [151]	VV-WBV f: 35 - 40Hz A: 1.7 - 2.5mm a: 2.2 - 5g \ddagger	5 weeks 3 sessions/week 9 - 18 mins/session	Multiple static and dynamic exercises	Sprint-trained $\sigma\varphi$ Age: 21yrs 10 X WBV 10 X Conventional training*	a) ISO and DYN knee strength b) CMJ c) Sprint	No change in ISO or DYN knee flexion or extension strength, knee extension velocity, sprint velocity, or CMJ.
Mulder <i>et al.</i> [67]	SA-WBV f: NR A: NR a: NR	8 weeks 12 sessions/week > 1 min/session	Static squat with added resistance (BRS)	Non-athletic σ Age: 33yrs 10 X WBV 10 X No intervention	a) ISO knee strength b) Muscle CSA	Quadriceps CSA: WBV (-3.5% \ddagger); No Intervention (-14.1% \ddagger). Difference between these groups significant. Quadriceps maximum torque: WBV (0%); No intervention (-16.8% \ddagger). No change in maximum voluntary activation levels.
Blottner <i>et al.</i> [69]	SA-WBV f: 19 - 25Hz A: 2.5 - 5.0mm a: 2.5 - 8g \ddagger	8 weeks 11 sessions/week 6 mins/session	Static squat with resistance (BRS)	Non-athletic σ Age: 33yrs 10 X WBV 10 X No intervention	A) Fiber phenotype b) MVC	ISO triceps surae force production: WBV (-9.2% \ddagger); No intervention (-32.9%). Difference between these groups significant. Myofiber type largely preserved in WBV group with soleus muscle showing a shift towards Type I fiber type.
Cheung <i>et al.</i> [154]	SA-WBV f: 20Hz A: NR a: NR	12 weeks 3 sessions/week 3 mins/session	Standing	Non-athletic φ Age: 72yrs 45 X WBV 24 X No intervention	a) Balance	Improved balance in WBV group only.
Bogaerts <i>et al.</i> [157]	VV-WBV f: 35 - 40Hz A: 1.25 - 2.5mm a: 4.4 - 11.4g \ddagger	52 weeks 3 sessions/week 2 - 15 mins/session	Static and dynamic squats	Non-athletic $\sigma\varphi$ Age: 68yrs 31 X WBV 30 X FIT training* 36 X No intervention	a) Muscle CSA b) CMJ c) ISO knee strength	ISO knee extension strength: WBV (9.8% \ddagger); FIT (13.1% \ddagger). CMJ: WBV (10.9% \ddagger); FIT (9.8% \ddagger). Muscle mass: WBV (3.4% \ddagger); FIT (3.8% \ddagger). NS difference between WBV and FIT groups. No intervention showed no training effect.
Bogaerts <i>et al.</i> [159]	VV-WBV f: 35 - 40Hz A: 1.25 - 5.0mm a: 4.4 - 11.4g \ddagger	52 weeks 3 sessions/week 2 - 15 mins/session	Static and dynamic squats	Non-athletic σ Age: 68yrs 31 X WBV 30 X Fitness programme* 36 X No intervention	a) Balance	WBV and FIT associated with a reduction in the number of falls and a NS but positive improvement in balance
Mulder <i>et al.</i> [161]	SA-WBV f: 16 - 26Hz A: 4mm a: 3 - 7.5g \ddagger	8 weeks 3 sessions/week > 3 mins/session	Multiple static and dynamic exercises with resistance (BRS)	Non-athletic σ Age: 32yrs 7 X WBV 7 X Intervention only 9 X No intervention	a) MVC b) Muscle CSA	Quadriceps CSA: No intervention (-13.5% \ddagger); unchanged in other groups. ISO knee extension strength: No intervention (-21.3% \ddagger); unchanged in other groups. Triceps surae CSA: No intervention (-28.3% \ddagger); Intervention only (-10.7% \ddagger); WBV (-11% \ddagger). Triceps surae MVC: No intervention (-24.9% \ddagger); Intervention only (-14.8% \ddagger); WBV (-12.3% \ddagger).
Machado <i>et al.</i> [155]	VV-WBV f: 20 - 40Hz A: 2 - 4mm 3 - 18g \ddagger	10 weeks 3 - 5 sessions/week 7.5 - 22 mins/session	Static squats	Non-athletic φ Age: 78yrs 14 X No intervention 15 X WBV	a) Muscle CSA b) RM c) Time up-go	CSA: WBV (Vastus medialis (8.7% \ddagger); Biceps femoris (15.5% \ddagger); Vastus lateralis (2.5% \ddagger); No intervention (0.3 - 5%). RM: WBV (38% \ddagger). Mobility: WBV (-9%). Muscle power: No change post-WBV and decrease post-no intervention.
Carson <i>et al.</i> [144]	VV-WBV f: 30Hz A: 2.5mm a: 6g \ddagger	4 weeks 3 sessions/week 1.5 mins/session	Seated heel-lifts with resistance	Non-athletic $\sigma\varphi$ Age 33yrs 9 X WBV 9 X Intervention only	a) RM b) MVC c) CMJH	RM: WBV (16% \ddagger); No intervention (16% \ddagger). MVC: WBV (14.7% \ddagger); No intervention (12.5%). CMJ: WBV (0.3% \ddagger); No intervention (0.7% \ddagger). No significant difference between groups. No change in rate of force development.
Petit <i>et al.</i> [145]	VV-WBV f: 30 - 50Hz A: 1 - 2mm a: 2.5 - 14g \ddagger	6 weeks 3 sessions/week 10 mins/session	Static squats	Non-athletic σ Age: 21yrs 12 X WBV (14g) 10 X WBV (2.5g) 10 X No intervention	a) DYN Knee strength b) MFGC c) SJ d) CMJ	DYN knee extension torque: WBV (14g) (13.2 - 16.3% \ddagger). SJ: WBV (14g) (4.8% \ddagger). CMJ: WBV (14g) (9.7% \ddagger). Significant difference between WBV groups. No change in MFGC.

Table 3.4: Chronic changes associated with whole body vibration in general populations with no neurological deficit (continued). VV: Vertically vibrating; SA: Side alternating; WBV: Whole body vibration; f: Frequency; A: Amplitude; a: a_{RMS} ; ♂: Male; ♀: Female; Age: Mean age of test population; CMJ: Counter movement jump; SJ: Squat jump; RM: Repetition maximum; ISO: Isometric; DYN: Dynamic; BMD: Bone mineral density; MVC: Maximum voluntary contraction; MFGC: Electrically elicited maximal force generating capacity; FFM: Fat free mass; RES: Resistance; BRS: Bed rest study; NS: Not statistically significant; MVJ: Multiple Vertical Jumps; †: Statistically significant; NR: Not reported; *: See original reference; ‡: Numerically estimated.

Author	Vibration param.	Training param.	Intervention	Test Population	Test	Results
Kern <i>et al.</i> [158]	NR-WBV f: 1 - 15Hz A: 1.5mm a: 0.05 - 1g‡	8 weeks NR sessions/week NR mins/session	Seated with 1Hz and 15hz applied to contralateral leg	Athletic ♂ Age: 69yrs 7 X WBV	a) Iso knee strength	ISO knee extension strength: 21 - 67%†increase found across both frequencies with no significant differences between frequencies found.
Osawa <i>et al.</i> [146]	VV-WBV f: 30 - 40Hz A: 2mm a: 2.5 - 4.5g‡	12 weeks 2 sessions/week 25 mins/session‡	Multiple static and dynamic exercises	Non-athletic ♂♀ Age: 27yrs 10 X WBV 8 X Intervention only	a) FFM b) ISO knee strength	ISO knee extension: WBV (10.9%); Intervention only (8.5%). ISO knee flexion: WBV (22.0%); Intervention only (24.3%). NS difference between groups. No change in FFM.
Item <i>et al.</i> [147]	SA-WBV f: 30Hz A: NR a: NR	5 weeks 3 sessions/week >12mins/session	Static squats with resistance and vascular occlusion	Non-athletic ♀ Age: 24yrs 12 X WBV 9 X No intervention	a) Respiration b) ISO knee strength c) Fiber phenotype d) Cycling power e) CMJH	Fiber pheontype: WBV (Type I (16.7%†), Type II (13.8%†), capillarisation (14%†)). Lean thigh mass: WBV (14%†). Maximal cycling power: WBV (8.7%†). Maximal cardiac output: WBV (8.9%†). No change in ISO knee extension strength, power, CMJ, VO2max. No intervention had no training effect.
von Stengel <i>et al.</i> [160]	VV-WBV f: 25 - 35Hz A: 1.7 - 2.0g 3 - 7g‡	72 weeks 2 sessions/week 6 mins/session	Static and dynamic squats and heel-lifts	Non-athletic ♀ Age: 69yrs 50 X Light Intervention* 51 X Intervention only 51 X WBV	a) Body fat b) ISO trunk and knee strength c) CMJ	Trunk extension strength: WBV (6.2%†); Intervention only (6.75%†). ISO knee extension strength: WBV (5%†). Trunk flexion strength: WBV (16.5%†). Body fat: Light intervention (-2.8%†); Intervention only (-11.4%†); WBV (-12.1%†). CMJ: Light intervention (0.4%); Intervention only (0.8%); WBV (1.7%). NS difference between groups.
Artero <i>et al.</i> [148]	VV-WBV f: 20 - 40Hz A: 1.25 - 2.5mm a: 1.41 - 11.3g	8 weeks 2 sessions/week 6 - 10 mins /session	Static and dynamic squats and jumping exercises	Non-athletic ♂♀ Age: 22yrs 15 X WBV 15 X Intervention only	a) 3RM b) DYN knee strength c) CMJ d) BMI e) Body fat	Muscle mass: WBV (3.4%†); Intervention only (4.5%†). 3RM: WBV(43%†); Intervention only (52%†). NS difference between groups. No change in DYN knee extension strength, CMJ. Body fat: WBV (-2.1%†).
Couto <i>et al.</i> [149]	VV-WBV f: 8 - 26Hz A: 3mm a: 0.5 - 5.7g‡	4 weeks 3 sessions/week >1 min/session	RM	Non-athletic ♂ Age: 26yrs 13 X Intervention only 14 X WBV (8Hz) 14 X WBV (26Hz) 14 X No intervention	a) MVC b) SJ c) CMJ d) MVJ e) Sprint	MVC: WBV (8Hz) (23.2%†); WBV (26Hz) (22.2%); Intervention (12.1%†). SJ: WBV (8Hz) (11.1%); WBV (26Hz) (9.6%). CMJ: WBV (8Hz) (8.7%†); WBV (26Hz) (7.5%). Significant difference between WBV and intervention only condition. NS difference between WBV conditions. NS changes in sptint speed of MVJ.

Muscle Strength

As described above, the outcome measures most commonly used to describe muscle strength include: unilateral or bilateral knee extension torque, unilateral knee flexion torque, trunk flexion and extension torque, maximum voluntary contraction, maximum electrically elicited force generating capacity, and repetition maximum. In the review below please refer to Table 3.2 - 3.4 for the specific tests used when the term strength is used. Strength increases of 3.7 - 67.0% were found after a period of WBV training [139,149,155,158]. However, others have shown no improvement [141, 143–145, 147, 148], or no statistically significant improvement when compared to a control group that carried out similar training paradigms in the absence of vibration, or conventional training programmes [140, 150, 151, 153].

An 8 to 16 week period of WBV training in a group of non-athletic, young men and women resulted in a 5.7 and 6.7% strength improvement for each group respectively. The control group, that did not perform any form of training, showed improvements of 2.2 and 5.2% for the same period. Nonetheless, a statistically significant improvement of 3.7% was shown for the WBV group when compared to the controls after 8 weeks of training [139]. A follow up study that investigated the effect of 32 weeks of WBV training showed a 9.8% improvement in strength, compared to a 7.8% improvement in controls that did not perform any exercise. This resulted in a non statistically significant between group difference of 1.9% [124]. The authors suggested that the lack of statistical significance at 16 and 32 weeks was due to the fact that most of the strength improvement occurred within the first 8 weeks of training, with a plateau thereafter, and that the improvements in muscle strength found for the controls was due to a learning effect over the course of the study [139]. While it is unclear whether the improvements were due to WBV or the exercise intervention alone, it was concluded that the intervention in the absence of vibration was of insufficient intensity to fully account for the strength improvements and must therefore be a consequence of the vibration therapy.

A similar study carried out for 12 weeks, but with a larger dose of vibration per session found strength improvements of 10.9 - 22.0% in the WBV group, and 8.5 - 24.3% in the controls that carried out the same intervention without vibration [146]. In both instances there was a non-significant difference between the improvements found for the vibration and control groups. Similarly, Carson *et al.* [144] found that WBV did not lead to an additional improvement in plantar flexor strength or one-repetition maximum when compared to participants that carried out the same intervention without vibration. These results suggest that the addition WBV to a training paradigm has a negligible effect on lower limb strength.

Delecluse *et al.* [140] compared a 12 week WBV training programme in a group of non-athletic, young females with three control groups: (i) a group that did not perform any exercise; (ii) a sham group that performed the same intervention, but in the absence of vibration; and (iii) a group that performed a conventional, resistance based strength training programme. They found that WBV training and the resistance programme each resulted in a comparable and statistically significant increase in strength of 13.6% and 14.4% respectively. A similar study found that a 24 week WBV training programme in a group of non-athletic, elderly females resulted in a 12% strength increase, and a resistance programme resulted in a 16% strength increase [153]. In each of these studies, the sham vibration condition and

no intervention condition did not result in any strength improvements, and no statistically significant difference was found when comparing WBV to the resistance training programmes.

A 5 week WBV training programme resulted in a significant and comparable increase in the one-repetition maximum of moderately strength-trained men when compared to those who carried out the same training programme in the absence of vibration [150]. Increases of 32.4% and 24.2% were found for the WBV and non-vibration programmes respectively. It was concluded that during one-repetition maximum, complete voluntary activation of the muscle was being achieved. WBV therefore facilitated the recruitment of additional muscle fibers. These additional fibers were activated by reflex activity, thus causing a larger increase in strength in the WBV group. Conversely, Delecluse *et al.* [151] compared WBV with a conventional training programme over a 5 week period in young, sprint-trained athletes. No strength increases were found and it was concluded that this was due to the small potential for performance improvement in previously trained athletes.

A similar study, but carried out over a much longer 52 week period, with a group of non-athletic, elderly women resulted in a statistically significant strength increase of 9.8% after the WBV training intervention [157]. This was comparable to an increase of 13.1% for the control group who carried out the conventional fitness programme over the same period. Von Stengel *et al.* [160] found comparable increments in muscle strength of 6.2%, in a similar population after 72 weeks of training. However, these results were not significantly different to the 6.75% strength increase found for the controls who carried out the same intervention but in the absence of vibration. Kern *et al.* [158] found that an 8-week period of WBV-training in athletic, elderly men resulted in a 21 - 67% increase in strength. This slightly unconventional study applied a different frequency of vibration to the contralateral lower-limbs. No significant difference was found for the leg trained at 1Hz compared to the other trained at 15Hz. Couto *et al.* [149] found that a 4-week WBV programme resulted in a 22.2 - 23.2% increase in strength. No difference was found between training at 8Hz or 26Hz, however these improvements were significantly larger than the control group who carried out the same intervention in the absence of vibration.

A meta-analysis carried out by Marin and Rhea [162] concluded that: (i) a period of WBV-training is more effective in untrained rather than athletic populations; (ii) the training effect is larger in elderly populations; (iii) strength improvements are larger if dynamic exercises with additional load are performed with vibration; and (iv) the training effect increases with frequency, amplitude, or dose of vibration. However, a review by Nordlund and Thorstensson [164] suggests that such strength improvements are largely attributable to a general training effect rather than WBV. They concluded that muscle hypertrophy and strength improvements are unlikely to occur because of the relatively low loads imposed on the muscle-tendon unit during WBV.

Muscle Power

The countermovement jump and the squat jump are the most common measures used to assess changes in muscle power during WBV training programmes. Other measures include isokinetic dynamometry and the rate of change of force production. A period of

WBV training was associated with a 0.3 - 12% increase in countermovement jump height [111, 139, 140, 144, 145, 153], and a 4.3 - 11.1% increase in squat jump height [142, 149]. However, as discussed in relation to changes in muscle strength, some studies have found no change [141, 144, 147, 148, 151], or no significant difference in these parameters when compared to controls who carried out the same intervention or conventional training programmes [142].

24 weeks of WBV training in a population of elderly women resulted in a 4.7% increase in muscle power, and a 4.9% increase in force velocity as assessed by a countermovement jump [152]. These increases were significant when compared to controls that did not participate in any form of regular exercise. However, in this study as with others, it is not clear if these improvements in muscle power were due to WBV or the intervention alone [111, 139, 152].

Several studies have compared the effect of a muscle training intervention carried out with and without WBV. DeRuiter *et al.* [141] did not find a significant increase in muscle power in a population of non-athletic, young men, though they did note that there was an increased tendency for rate of force production in the WBV group. Similar findings are reported by Carson *et al.* [144]. Conversely, Petit *et al.* [145] found that a high-frequency (50Hz) WBV training programme resulted in a significantly larger increase in jump height when compared to a low-frequency (30Hz) training programme. These findings are in part supported by Couto *et al.* [149], who found that WBV elicited a 9.6 - 11.1% increase in squat jump height, and a 7.5 - 8.7% increase in countermovement jump height, in a population of young, non-athletic men. However, no significant difference was identified when comparing those who trained at the different frequencies of vibration. It is possible that the lack of significant difference is due to the different range of frequencies tested (6 & 26Hz), compared to those described above (30 & 50Hz).

Other study designs have compared a WBV training programme with conventional resistance based training programmes. Delecluse *et al.* [140] found that WBV was more effective than the conventional programme in a population of non-athletic young women, where a 7.6% increase in countermovement jump height was identified. Similar improvements in a non-athletic elderly populations of 1.7 - 12.0% have been reported, though these improvements were not significantly different to those who carried out conventional maintenance, resistance, or fitness training, who similarly showed increases in countermovement jump height of 0.4 - 16.0% [153, 157, 160]. Ronnestad [150] identified a 9.1% increase in countermovement jump height in a population of moderately strength-trained young men, though this was not significantly different to the improvements of 4.2% found for the control group.

A meta-analysis by Marin and Rhea [165] concluded that: (i) overall WBV could be used to increase muscle power; (ii) WBV training is more effective in older rather than younger populations; and (iii) the best improvements in power are achieved by training between 35 - 40Hz, increasing amplitude, or applying a dose of vibration of 360 - 720 seconds in duration. In addition, it has been suggested that the lack of statistical significance of reported findings may be due to a lack of power of the statistical tests used and the typically small number of participants recruited for study [141].

Counteracting Neuromuscular Decline

A series of bed-rest studies have investigated the potential of WBV training as an exercise modality to counteract muscular decline [67,69,161]. Physiological decline during prolonged bed-rest is well documented [63], and is now used as a model of disuse during spaceflight [66,71]. 8 weeks of bed rest in young, healthy, non-athletic adults resulted in a 13.5 - 14.1% reduction in quadriceps cross sectional area [67,67], and a 28.3% reduction in the calf cross sectional area [69]. A corresponding 16.8 - 21.3% reduction in knee extensor torque [67,67], and a 24.9 - 32.9% reduction in the isometric force production of the triceps surae was found in the same population [69,161]. In addition, a significant increase in the proportion of fast twitch (Type II) myofibers was found in the soleus muscle [69]. 8 weeks of WBV training was then shown to counteract some of these trends and therefore be effective in preserving muscle size and function [67,69]. A smaller reduction in muscle cross sectional area of 3.5% and 11% was found for the quadriceps and triceps surae respectively. Knee extension torque was preserved, and a smaller reduction of 9.2% in triceps surae isometric force production was found. A shift towards the slow-twitch (Type I) myofiber type was found in the soleus muscles of those subjects trained with WBV. However, in the final study of the series, Mulder *et al.* [161] were unable to identify a significant difference in response between those that trained with WBV, and those who carried out the same exercise paradigm but in the absence of vibration.

Other parameters

No change in agility, postural sway, sprint speed, or upper limb grip strength has been found in young, athletic and non-athletic adults [14,124,139,142]. More promising findings have been identified in elderly populations, where WBV has been associated with improvements in balance and a reduction in the number of falls [154,159]. As described in Section 3.5.1, the metabolic demand of WBV was moderate, where oxygen uptake during WBV was 50% of maximum [113]. In addition to this finding, Item *et al.* [147] found that WBV training did not change $\dot{V}O_{2max}$, but did lead to an increase in maximal cycling power, muscle capillarisation, and lean thigh mass. A 2.2% increase in fat-free mass, and a 2.1% reduction in body fat has been associated with WBV training [143,148], though these findings have not been replicated by others [146].

3.6.2 Neurologically Compromised Populations

Chronic physiological adaptations after a period of WBV training have been investigated in populations that suffer from a neurological deficit. These populations include: stroke [166,167], multiple sclerosis [168–171], and spinal cord injury [172,173]. Typical outcome measures from these studies include: (i) balance [166,167,169–171]; (ii) timed up-and-go [167–171]; (iii) muscle tone/spasticity [167,168,173]; (iv) walking [166–172]; and muscle strength [167–169,171].

These studies have most commonly been carried out in community dwelling populations, whose disease or injury occurred greater than one year prior to the beginning of the study. Training programmes have been carried out for 3 - 10 weeks, with training carried out 2 - 5 times per week. The frequencies and amplitudes typically used are 25 - 50Hz, and 1 - 4mm

respectively. The total dose of vibration per session is typically between 3 - 15 minutes per session, and overload is achieved by increasing frequency, amplitude, or dose of vibration, or by decreasing the duration of periods of rest. Participants almost exclusively perform static or dynamic squats, heel-lifts, or lunges without additional weight.

As with the studies reviewed for the general population, some studies have shown WBV training has a positive effect, for example on walking in a population suffering from multiple sclerosis [170]. However, others have been unable to show any improvement [168], or a significant difference when compared to controls who carried out the same intervention during a sham vibration condition [171]. The original research articles reviewed in this section are summarised in Table 3.5, and the results are discussed in greater detail in later sections.

Table 3.5: Chronic changes associated with whole body vibration in populations with a neurological deficit. Deficit duration: Time since stroke, spinal cord injury, or onset of multiple sclerosis; VV: Vertically vibrating; SA: Side alternating; WBV: Whole body vibration; f: Frequency; A: Amplitude; a: a_{RMS} ; ♂: Male; ♀: Female; Age: Mean age of test population; ISO: Isometric; DYN: Dynamic; NS: Not statistically significant; †: Statistically significant; NR: Not reported; ‡: Numerically estimated.

Author	Vibration param.	Training param.	Intervention	Test Population	Test	Results
Van Nes <i>et al.</i> [166]	SA-WBV f: 30Hz A: 1.5mm a: 3.8g†	6 weeks 5 sessions/week 3 mins/session	Static squats	Stroke ♂♀ Deficit duration: 5wks Age: 61yrs 27 X WBV 26 X Exercise with music	a) Balance b) Mobility c) Trunk control d) Motricity index e) Activities of daily living	Improvement in balance (70%†); mobility (64%†); trunk control (14%†); activities of daily living (49%†); and motricity index (26%†). NS difference between WBV and control groups. Larger improvements during intervention period than post-intervention period. Conclude WBV has same treatment effect as exercise with music.
Schyns <i>et al.</i> [168]	VV-WBV f: 40Hz A: 1mm a: 4.6g†	4 weeks 3 sessions/week 5 mins/session	NR	Multiple sclerosis ♂♀ Deficit duration: 6.7yrs Age: 48yrs 12 X Randomised cross-over trial.	a) Muscle tone b) ISO muscle force c) Sensation d) Proprioception e) 10m walk test f) Time up and go	Tendency for increased tone in absence of WBV therapy. NS increase in ISO muscle force production of knee, hip, and ankle flexors and extensors. NS improvement in 10m walk test and time up and go. No change in sensation or proprioception. NS difference between groups. Conclude tests underpowered to detect between group differences.
Ness <i>et al.</i> [172]	VV-WBV f: 50Hz A: 1 - 2mm a: 7.1 - 14.2g†	4 weeks 3 days/week 3 mins/session	Static squats	SCI ♂♀ Deficit duration: >1yr Age: 48yrs 17 X WBV	a) Walking	Improved walking speed (24%†); cadence (10%†); step length (18 - 19%†); and limb co-ordination (10 - 13%†).
Ness <i>et al.</i> [173]	VV-WBV f: 50Hz A: 1 - 2mm a: 7.1 - 14.2g†	4 weeks 3 days/week 3 mins/session	Static squats	SCI ♂♀ Deficit duration: >1yr Age: 51yrs 16 X WBV	a) Spasticity	Significant reduction of first swing excursion (20%†) as assessed during a pendulum test. NS change in the number of oscillations. NS difference in response between those that use anti-spastic medication, and those that do not.
Broekmans <i>et al.</i> [169]	VV-WBV f: 25 - 45Hz A: 2.5mm a: 4.4 - 14.4g†	20 weeks 2 - 3 sessions/week 2.5 - 16.5 mins/session	Static and dynamic squats	Multiple sclerosis ♂♀ Deficit duration: NR Age: 48yrs 11 X WBV 12 X No intervention	a) ISO & DYN strength b) Speed of movement c) Balance d) Timed up and go e) 2 min walk test f) Timed walk test	ISO and DYN knee flexion and extension strength (-8.9 - 6.8%); NS change in speed of movement; Balance (-6.7%); Time up and go (-4.4%); 2 min walk test (6.8%); and timed walk test (-3.5%). NS difference between WBV group and no-intervention group.
Mason <i>et al.</i> [170]	SA-WBV f: 15 - 25Hz A: 1.30 - 3.65mm a: 0.83 - 3.65g†	8 weeks 3 sessions/week 5 - 8 mins/session	Static squats	Multiple sclerosis ♂♀ Deficit duration: 10.9yrs Age: 50yrs 15 X WBV	a) Quality of life b) Timed up and go c) Functional reach test d) Balance e) Time 10m walk test	Improved physical functioning (19%†) after 4 weeks; Faster timed up and go (11%†), 10m walk test (13%†), and balance (15%†) compared to baseline. NS change in functional reach.
Claerbout <i>et al.</i> [171]	VV-WBV f: 30 - 40Hz A: 0.8mm a: 2.7g @ 40Hz	3 weeks 3 - 4 sessions/week 7 - 13 mins/session	Static and dynamic squats, heel-lifts, and lunges	Multiple sclerosis ♂♀ Deficit duration: 11.6yrs Age: 43.5yrs 16 X WBV 14 X Damped WBV 17 X No WBV	a) ISO strength b) 3min walk test c) Timed up and go d) Balance	ISO: WBV (34.6 - 56.2%), Damped WBV (19.5 - 34.5%), No WBV (2.8 - 16.5%). Changes in ISO strength of quadriceps and hamstring significant; NS improvements in all other measures: Timed up and go: WBV (9.1%), Damped WBV (16.2%), No WBV (4.9%); 3min walk test: WBV (38.7%), Damped WBV (31.8%), No WBV (15.8%); Balance: WBV (10%), Damped WBV (12%), No WBV (3.9%).
Brogardh <i>et al.</i> [167]	VV-WBV f: 25Hz A: 0.1 & 1.9mm a: 0.2 & 3.4g†	6 weeks 2 sessions/week 3 - 12 mins/session	Static squats	Stroke ♂♀ Deficit duration: 2.9yrs Age: 62.7yrs 16 X WBV (A = 1.9mm) 15 X CTRL (A = 0.1mm)	a) ISO & DYN strength b) Balance c) Muscle tone d) Timed up and go e) Gait speed f) 6min walk test	Timed up and go: WBV (8%†), CTRL (6%†); 6min walk test: WBV (5%†), CTRL (6%†); Balance: WBV (4%†); Gait speed: WBV (5%†); ISO strength in paretic limb: CTRL (12%†). NS difference between groups for any outcome measure.

Stroke

A 6 week period of WBV training in an acute stroke population resulted in a significant improvement in balance, mobility, trunk control and activities of daily living [166]. However, these improvements were not found to be significant when compared to a control group that carried out same exercises with music, suggesting that WBV does not infer any additional benefit to exercise alone during the early stages of stroke recovery. In addition, it is unclear to what extent the improvements observed would have occurred naturally in the ordinary course of recovery. A similar study carried out with a chronic stroke population identified smaller improvements, in addition to increases in isometric strength of the paretic limb [167]. In each study, the small and non-significant differences found when compared to the control groups were attributed to the low intensity of the vibration therapy, and thus the small demand it exerted on the neuromuscular system [166,167].

Multiple Sclerosis

An 8 week WBV training programme resulted in a significant improvement in the balance, walking capabilities, and timed up-and-go of community dwelling individuals with multiple sclerosis when compared to baseline [170]. However, these results were not replicated when compared to others who investigated the same parameters but using a study of a randomised cross-over design [168], or when compared to a different control group [169]. Broekmans *et al.* [169] concluded that the poor results found in their study might be a consequence of overtraining, as the frequency, amplitude and dose of vibration exceeded those of other studies [168,170]. On a more positive note though, Schyns *et al.* [168] report an increased tendency of muscle tone in those who did not perform WBV training. This is supported by similar finding in SCI [173]. In a similar and well controlled study by Claerbout *et al.* [171], larger improvements in balance, walking capabilities and timed up-and-go were identified, in addition to significant increases in quadriceps and hamstring isometric strength. The authors concluded that the poor results presented in previous studies were due to the fact that the participants were only mildly affected by their neurological impairment, and were already functioning near the peak of the physical capability, therefore presenting small scope for additional improvement. In contrast they recruited a population that was severely affected by disability, and thus had more potential for improvement with training [171].

Spinal Cord Injury

Literature describing the effect of WBV training in SCI is limited. Ness *et al.* [172,173] investigated the effect of WBV training on walking and spasticity in a population of chronic (>1 year), motor-incomplete SCI individuals. 4 weeks of WBV training improved walking speed (24%), cadence (10%), step length (18 - 19%), and co-ordination (10 - 13%), when compared to baseline measures [172]. Subjects used as their own controls in this way was justified because these parameters were assumed to have reached a plateau within one year of cord injury. In addition, a decrease in first swing excursion during a pendulum test (20%) was found, indicating a significant decrease in quadriceps spasticity [173]. Furthermore, improvements in tone were independent of the prescription of anti-spastic medication, and no significant difference was identified between those taking such medication, and those not taking it. Analysis showed that the largest improvements were found for those with the

highest degree of spasticity at the beginning of the study [173].

3.7 Summary of Literature

WBV as a training modality has been reasonably well described for a wide range of sample populations with no neurological deficit. Depending on the specific aim of the exercise programme, the training effect of WBV tends to increase with frequency, amplitude, or dose of vibration [130, 145, 149, 162, 165]. However, relatively few studies have directly compared the acute neuromuscular response of different vibration conditions, with a view to validating their application in a training programme [72, 73, 174]. Furthermore, no such evidence exists for the SCI population.

It has also been shown that WBV training may be of most benefit to those in the general population who are un-trained or elderly [162]. The hypothesis is that such populations possess a greater capacity for improvement because they have not been previously performing to the peak of their ability. While it can be argued that this hypothesis is not limited solely to WBV training programmes, larger improvements have been reported for populations more severely affected by neurological injury or disease [171, 173]. It was therefore concluded that WBV may be particularly suited to those with a severe neurological deficit such as SCI. However, studies describing the acute response of such populations are limited, particularly for those with an acute and sub-acute injury.

SA- and VV-WBV are the most commonly investigated systems. Vibration has almost exclusively been applied by these systems with the participant in an upright or standing position, and sometimes carrying additional load. This set-up therefore precludes the participation of those unable to stand or support their own body weight. It is most likely for this reason that the majority of research studies investigating WBV have been carried out in the general population, or with those only moderately affected by neurological de-conditioning or paralysis. More recently, some investigators have applied WBV in other ways with mixed success. Vibration was successfully shown to attenuate neuromuscular decline, when applied to subjects in a supine position during a series of bed-rest studies [67, 69, 161]. However, in these studies the participants were provided with strapping over the shoulders that inferred additional load, making the exercise more analogous to standing, and the exercise was thus termed resistive vibration exercise. Others have applied WBV in a seated position [87], or on a tilt-table [115] in a sample of motor complete SCI subjects. A poor neuromuscular response was found for both conditions, though an increase in blood flow was identified on the tilt-table [115]. Likely reasons for the small response on the tilt-table include: the chronicity of the cord injury [115] and the poor transmission of vibration to the body [137]. Better transmission of vibration was shown for SCI subjects bearing their full weight in a standing position with the support of a conventional standing frame [138]. WBV applied in this way was shown to have a short term influence on the H-reflex, though the general neuromuscular response during vibration was not reported [117]. The neuromuscular response of the sub-acute, motor complete and motor incomplete SCI population, during WBV applied in the conventional upright position has yet to be investigated.

It remains equivocal as to whether or not movement artefact is present in an EMG recorded during WBV. Several authors have put forward evidence suggesting the presence of artefact and advocating its removal [75,107,126], meanwhile others have suggested that the influence of artefact is minimal and that the additional myoelectric signals manifested in the EMG are stretch reflexes that are occurring in synchrony with the vibrating platform [88]. It remains plausible that a stretch reflex may be evident in an EMG recorded during WBV because there is firm evidence that the stretch receptor plays a central role in the neuromuscular response to WBV [88]. However, notwithstanding the potential presence of such stretch reflexes, it is not possible to preclude the presence of movement artefact in the signal either. The investigation of WBV with a specific subset of the SCI population may improve the understanding of this issue. Damage to the lower motor neuron results in areflexia, and therefore a total absence of the stretch reflex. Studying the acute neuromuscular response of this subset provides a unique opportunity to determine, and if present quantify, the movement artefact present in an EMG recorded during WBV.

It was thus concluded to investigate the neuromuscular response of both neurologically intact and SCI participants under the same experimental conditions by integrating a WBV platform with a partial Body Weight Support (pBWS) system.

3.7.1 Open Research Questions

The application of WBV in a standing position under conditions of reduced load, as permitted by reducing body weight using a pBWS system has not been investigated in a neurologically-intact or SCI population. As such is is not known how the reduction of load may affect the neuromuscular response to WBV.

The open research questions which were identified included: (i) can WBV be applied in an upright position to those who cannot support their own weight by providing pBWS? (ii) does WBV elicit a contraction in muscle when body weight is reduced? (iii) What is the relationship between vibration frequency, vibration amplitude, level of pBWS and the neuromuscular response? (iv) Are the same trends in neuromuscular response different for different levels of pBWS? (v) Is there a measurable neuromuscular response from those with an early-stage SCI to WBV, and if so, is there a relationship between the neuromuscular response and the vibration parameters? (vi) Is there a difference in response between those with no neurological deficit and those with SCI? (vii) How much movement artefact is present in an EMG recorded during WBV?

3.8 Aims & Objectives

The open research questions were addressed by formulating the following aim: measure and quantify the acute steady-state neuromuscular response of neurologically intact and SCI populations before, during and after WBV-pBWS.

A steady-state response was defined as a period of neuromuscular activity which did not fluctuate for a significant amount of time. A non steady-state response was defined as a neuromuscular response that fluctuated, for example a transient or inconsistent change which

typically occurred during the transition from a pre-Vibration to Vibration condition. This aim was achieved by addressing by the following technical and physiological objectives:

3.8.1 Technical Objectives

1. Integrate a WBV platform with a pBWS system.
2. Quantify the transmission of vibration from the WBV platform to the lower limbs, by quantifying vibration frequency, vibration amplitude and acceleration at key locations on the vibration platform and legs.
3. Estimate the proportion of vibration artefact in every EMG recorded during WBV.
4. Assess the suitability of passive vs active EMG electrodes for WBV applications.
5. Optimise the off-line filtering process for vibration artefact removal.
6. Optimise the data analysis process for classification of EMG and rejection of outlying data.

3.8.2 Physiological Objectives

1. Quantify the magnitude of the EMG response of the Tibialis Anterior (TA), Medial Gastrocnemius (M-GA), Lateral Gastrocnemius (L-GA), Vastus Lateralis (VL), Rectus Femoris (RF), and Biceps Femoris (BF) to WBV.
2. Describe the changes in EMG activity with respect to vibration frequency, vibration amplitude and level of pBWS.
3. Investigate the temporal relationship between the EMG and the position of the WBV platform.

Chapter 4

WBV Methods

A WBV platform was integrated with a pBWS system. pBWS was used facilitate standing in a patient population unable to support their own weight, such as SCI. This combined system is described hereafter as WBV-pBWS and is shown in Figure 4.1. The intention of the system was to allow those with a SCI to use WBV in a conventional manner.

Such a system has not been investigated previously. The acute neuromuscular response to WBV at reduced load was therefore not known. The initial investigation into WBV-pBWS was described with a general population with no neurological deficit. This population was used in the first instance to establish a normative baseline and to determine a suitable testing protocol for application in SCI. The system was then tested with a SCI population.

EMG was used to measure neuromuscular activity of key lower limb muscle groups before, during and after WBV. A range of different vibration frequencies, vibration amplitudes and levels of pBWS were tested in order to comprehensively describe the neuromuscular response to WBV. The EMG were analysed in the time and frequency domains with additional off-line filtering applied to take account of the unique challenges encountered while acquiring EMG signals during WBV. The findings of this study were used to inform the study carried out with a SCI population. This chapter outlines the experimental and analysis methods used for both studies.

4.1 Experimental Methods

This section outlines the experimental methods and outcome measures of this study. Where appropriate additional details are provided through worked examples and figures.

4.1.1 Equipment

Figure 4.1 outlines a general overview of the equipment used in this study. Vibration was applied using a SA-WBV device (Galileo Basic, Novotec Medical GmbH, Pforzheim, Germany), with the participant in a standing position. Body weight unloading was achieved using a pBWS system and an upper body harness (Loko S70, Woodway USA Inc, Waukesha, USA). Neuromuscular activity was recorded from a number of key leg muscles using two types of EMG amplifier: (i) an 8-channel Bagnoli Desktop EMG system (Delsys Inc., Massachusetts,

USA), and (ii) a NeuroLog System (Digitimer Limited, England, UK). Active EMG recording electrodes (DE-2.1, Delsys Inc., Massachusetts, USA) were used with the Delsys system, and passive EMG recording electrodes (Blue Sensor, Ambu A/S, Ballerup, Denmark) were used with the Neurolog system. Acceleration (ADXL 335, Analog Devices Inc., Massachusetts, USA) was recorded from key locations on the WBV platform and the participants body. All data was acquired at 1kHz, using a 16bit analog-to-digital converter (DAQCard-6036E, National Instruments Corporation, Texas, USA). Data acquisition was synchronised and processed using Matlab (MatlabR2010a, The Mathworks Inc., Massachusetts, USA), and stored on a laptop PC (Latitude D630, Dell, Texas, USA).

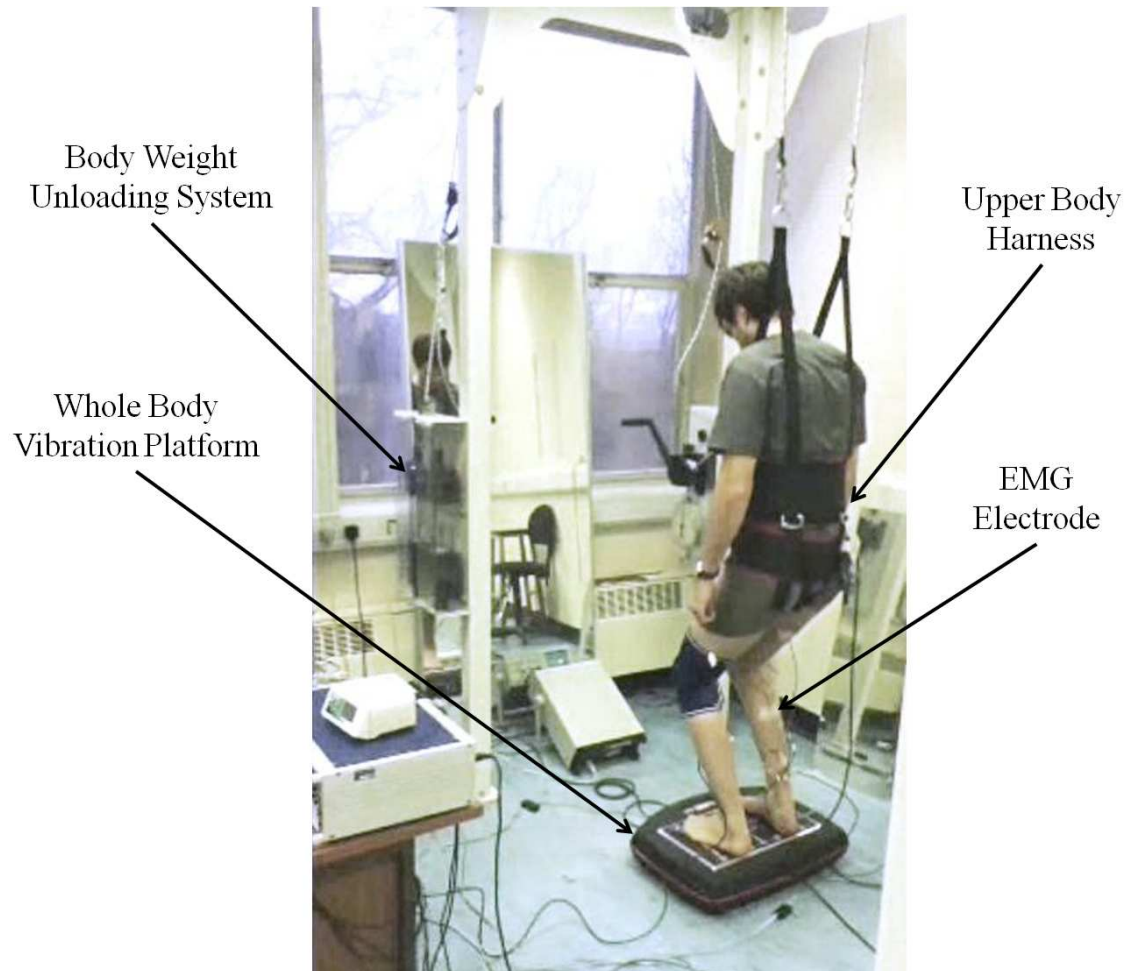


Figure 4.1: Picture describing experimental setup.

4.1.2 Participants

Neurologically Intact

Ten able-bodied volunteers (Table 4.1) were recruited to participate in this study. Exclusion criteria included: pregnancy, acute thrombosis, lower limb implants, acute inflammation of the locomotor system active orthosis or arthropathy, acute tendinopathy in the lower limbs, acute hernia, acute discopathy, gallstones or stones in the urinary tract collection system, rheumatoid arthritis, epilepsy, psychotic illness, neurotic disturbances or reduced cooperation.

Ethical approval (FBLS1014) was given by the Faculty of Biomedical and Life Sciences Ethics Committee, University of Glasgow. A copy of the research ethics approval letter is shown in the appendix. All participants gave written and informed consent prior to participation in this study.

Table 4.1: Summary of recruited able-bodied subjects. F = Female; M = Male.

Subject	Age [yrs]	Weight [kg]	Gender
S1 _{AB}	30	54	F
S2 _{AB}	26	70	M
S3 _{AB}	40	69	M
S4 _{AB}	26	80	M
S5 _{AB}	25	76	M
S6 _{AB}	26	50	F
S7 _{AB}	24	84	M
S8 _{AB}	23	89	M
S9 _{AB}	26	95	M
S10 _{AB}	44	69	F
Mean (SD)	29 (7)	73 (14)	-

SCI

Fourteen volunteers with a SCI were recruited from the Queen Elizabeth National Spinal Injuries Unit, Glasgow (Table 4.2). Similar inclusion and exclusion criteria were used with the following additions: (i) male or female volunteers with a traumatic SCI, (ii) motor complete or motor incomplete SCI, and (iii) a spinal fracture deemed stable for re-ambulation or rehabilitation. Additional exclusion criteria included: (i) the presence of pressure sores in the foot or trunk area, (ii) patients with a history of severe susceptibility to autonomic dysreflexia, and (iii) unstable fracture(s). Ethical approval was given by National Health Service West of Scotland Research Ethics Service (ref: 11/AL/0096). All participants gave written and informed consent prior to participation in this study. A copy of the research ethics approval letter is shown in the appendix.

Table 4.2: Summary of recruited SCI participants. F = Female; M = Male; AIS = ASIA Impairment Scale; ASIA: American Spinal Injury Association; Cx = Cervical Spinal Segment; Tx = Thoracic Spinal Segment; Lx = Lumbar Spinal Segment; x = Spinal Vertebral Number; TSI = Time Since Injury; SD = Standard Deviation; IQR = Interquartile Range.

Subject	Age [yrs]	Weight [kg]	Gender	AIS	Level	TSI [days]
S11 _{SCI}	19	67	M	B	L3	107
S12 _{SCI}	31	67	M	A	C4	189
S13 _{SCI}	60	85	M	D	T5	52
S14 _{SCI}	49	85	M	C	C7	169
S15 _{SCI}	48	77	M	C	C6	179
S16 _{SCI}	57	74	M	D	C4	52
S17 _{SCI}	28	65	M	B	T7	5087
S18 _{SCI}	52	61	F	C	C5	51
S20 _{SCI}	58	80	M	D	C4	284
S21 _{SCI}	51	81	M	C	L3	41
S22 _{SCI}	17	60	M	A	T10	262
S23 _{SCI}	58	65	F	C	T2	97
S24 _{SCI}	56	87	M	D	C4	172
S25 _{SCI}	40	93	M	A	C4	223
Mean (SD)	45 (15)	75 (11)	-	-	-	498 (1323)
Median (IQR)	50 (24)	76 (19)	-	-	-	171 (151)

The mean and median values are shown in Table 4.2, including the outlying Time Since Injury (TSI) from Subject S17_{SCI}. If removed, the mean TSI for the grouped data reduced to 144 days with a standard deviation of 84 days. The results from this participant were expanded upon as part of case based discussion.

4.1.3 Experimental Setup

Neurologically Intact

Each neurologically intact subject participated in three experimental sessions. The participant was familiarised with the WBV device and measurement equipment during the first session. No data gathered during the familiarisation session was used for the subsequent offline analysis. Testing was carried out in the two subsequent sessions. The same experimental protocol was used in each testing session. The session began by preparing the subject for EMG measurement. Active EMG electrodes were placed on the TA, M-GA, VL, and BF of the subject's preferred leg. Passive electrodes were placed on the L-GA and RF of the same leg. Different muscles from the quadriceps and triceps surae muscle groups were selected for the active and passive electrodes to facilitate optimum placement of the electrode, rather than to compromise the quality of the EMG by positioning a second electrode at an inadequate point on the same muscle. This setup was determined to be appropriate because subsequent analysis focusses on the relative performance of each electrode with respect to the muscle group rather than the individual muscles within that group. The muscles were located by palpation and electrode placement was done according to Table 4.3. The EMG electrodes were setup in a bipolar configuration and aligned along the direction of the muscle fibers. The reference electrode was affixed to the lateral malleolus of the same leg. The MVC were performed in triplicate in the supine position. The supine position was adopted for each MVC such that the joint over which the muscle of interest acted was allowed to move through its

full range of motion. Resistance was provided by the experimenter during each MVC. Full range of movement of the joint was allowed to ensure that the maximum voluntary force generating capacity of the muscle was captured and not just that for one muscle length. A rest period of 60 - 120s was allowed between each maximum contraction. The maximum recorded value over the three MVCs was taken for subsequent analysis. The subject next donned an upper body harness for the pBWS system.

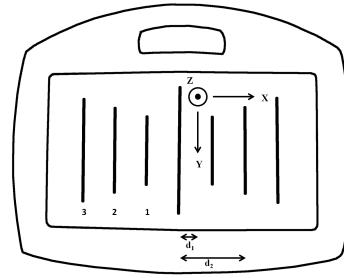
Table 4.3: Location of electrode placement.

Muscle	EMG Electrode Placement
Tibialis Anterior (TA)	Lateral of the tibial crest, 6cm distal of the tibial tuberosity
Medial Gastrocnemius (M-GA)	8cm distal of the medial femoral condyle
Lateral Gastrocnemius (L-GA)	On the lateral surface of the calf muscle group, 8cm distal of the popliteal crease
Vastus Lateralis (VL)	On the lateral surface of the quadriceps muscle group, 8cm caudally from the patella
Rectus Femoris (RF)	On the midpoint between the most caudal point of the patella and the anterior superior iliac spine
Biceps Femoris (BF)	On the midpoint between the head of the fibula and ischial tuberosity

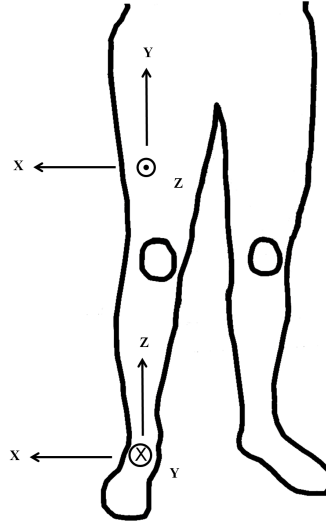
Platform acceleration was recorded, by affixing a triaxial accelerometer to the platform surface at a position located 42mm from the central axis. The method for measuring the position of the accelerometer is described in a later section. Acceleration was also recorded from the dorsal surface of the subjects' foot and from the EMG electrode placed on their VL. Each accelerometer was affixed to the body using hypoallergenic tape. The placement and orientation of the accelerometers are shown in Figure 4.2. The anatomical references relating the individual components of acceleration (x, y & z) to the directions of motion of the participant are described in Table 4.4.

Table 4.4: Anatomical references for acceleration measurements

Muscle	X-axis	Y-axis	Z-axis
Platform	Medial-Lateral	Anterior-Posterior	Longitudinal
Foot	Medial-Lateral	Anterior-Posterior	Longitudinal
Vastus Lateralis	Medial-Lateral	Longitudinal	Anterior-Posterior



(a) Vibration platform



(b) Axes relative to lower limbs

Figure 4.2: Schematic showing the location and positive orientations of the x-, y- and z-axes of the accelerometers placed on the WBV platform, foot and thigh. The x-, y- and z-axes were related to anatomical-axes in Table 4.4. d_1 = Displacement of accelerometer, along the medial-lateral axis, that has been affixed to the WBV platform. This displacement is estimated using Equation (4.1); d_2 = Position of foot on WBV platform and represented as a displacement along the medial-lateral axis. This position is prescribed as dictated by the experimental protocol.

SCI

Each SCI subject participated in two experimental sessions and the same experimental protocol was used in each session. The session began by preparing the subject for EMG measurement. Active¹ EMG electrodes were placed on the TA, M-GA, and VL² of both

¹Passive EMG recording electrodes were shown to be excessively susceptible to vibration induced motion artefact and therefore had a significantly compromised performance relative to active electrodes. They were not used with the SCI participants for this reason.

²The neuromuscular response of the BF was shown to be small relative to the other muscles during testing with the neurologically intact population. There was therefore a small likelihood of detecting a significant change in neuromuscular response from the SCI participants either. The BF was not tested in the SCI population for this reason.

legs³. The muscles were located by palpation and the electrodes were placed according Table 4.3. MVC were performed in triplicate where possible with the ISCI participants only. Each MVC was carried out in a seated position from the wheelchair. A rest period of 60 - 120s was allowed between each MVC. The joint over which the muscle of interest acted was allowed to move through its full range of motion during each MVC. Resistance was provided by the experimenter in the event that the participant had sufficient volitional strength to counteract the resistance. The maximum of the three recorded MVC's was taken for subsequent analysis.

The subject next donned an upper body harness for the pBWS system. Platform acceleration was recorded, by affixing an accelerometer to the platform surface at a perpendicular displacement of 58mm from the central axis. Acceleration was also recorded from the dorsal surface of the subjects left foot and from the electrode placed on the VL. Each accelerometer was affixed to the body using hypoallergenic tape.

4.1.4 Accelerometer Placement

Figure 4.3 outlines a schematic which describes how the displacement d_1 in Figure 4.2(a) was computed.

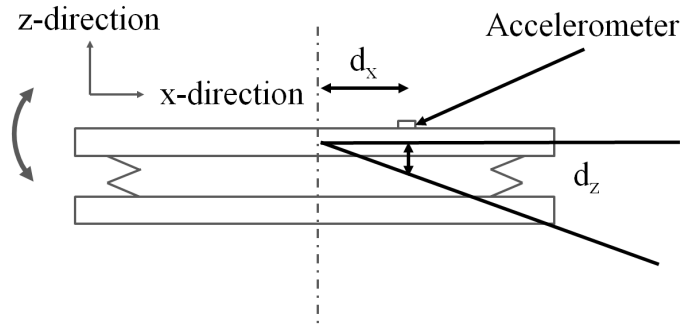


Figure 4.3: Schematic describing how acceleration, as recorded from the surface of the vibration platform may be used to estimate the point at which the accelerometer has been affixed to the vibration device.

Displacement in the z-direction (d_z) was estimated using the equation below, which is an expansion of Equation 3.1:

$$d_z = \frac{a_{peak}}{4\pi^2 f^2}$$

Where d_z = Displacement in z-direction; a_{peak} = Maximum acceleration measured in the z-direction using an accelerometer; and f = Frequency of vibration. When using an accelerometer a_{peak} and f are directly measured.

The location of the accelerometer (d_x) and displacement in the z-direction (d_z) are linearly related if it is assumed that the WBV platform is completely rigid. According to the manufacturer guidelines, a point on the platform that is located 55mm along the x-axis

³A bilateral imbalance in lower limb muscle strength is common in SCI and it was therefore concluded to be appropriate to measure the neuromuscular response of both legs.

equates to a displacement of 1mm in the z-direction (c.f. Figure 4.3). This linear relationship can be written as:

$$l_x = 55d_z$$

Where l_x = Location along the x-axis; and d_z = Displacement in the z-direction.

The location along the x-axis can therefore be calculated by:

$$l_x = \frac{55a_{peak}}{4\pi^2 f^2} \quad (4.1)$$

Accordingly, at a measured peak acceleration of 4.08m/s² and frequency of vibration of 11.66Hz, the location (l_x) at which the accelerometer was affixed to the surface of the WBV platform (d_1 (c.f. Figure 4.3)) was 42mm.

In addition, the acceleration of any point on the WBV platform may be extrapolated from a single acceleration measurement made from the platform surface. This extrapolated acceleration (a_E) was calculated by:

$$a_E = \frac{d_2}{d_1}a \quad (4.2)$$

Where a_E = Extrapolated acceleration; a = Recorded Acceleration; d_1 = Location of accelerometer along x-axis (c.f. Figure 4.2); d_2 = Location of point of interest along x-axis.

4.1.5 Experimental Testing

Testing was carried out at nine discrete vibration conditions, which included vibration frequencies of 12, 20 and 27Hz and vibration amplitudes of ± 1 , 2 and 3mm. Each combination of these vibration conditions was tested with 0, 50 and 100% pBWS provided to the neurologically intact participants yielding a total of 27 discrete test conditions per experimental session. The same vibration conditions were tested with 50% or 100% pBWS provided to the SCI participants depending on the severity of injury and the degree of the functional capability, therefore yielding a total of 9 discrete test conditions per experimental session. The order of testing was randomised. An individual test began with the subject taking a squat position on the vibration platform with the knees bent to 125° (i.e. 55° from straight leg position). Knee angle was verified using a manual goniometer and the head of the tibia was used as the reference point for joint articulation. The subject held this position for 60s and the test was carried out as outlined in Figure 4.4. A minimum rest period of 30s was allowed between each discrete vibration condition. Recorded data from each individual test was stored for subsequent off-line analysis.

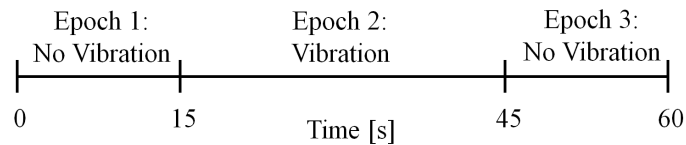


Figure 4.4: Time-line for each discrete test.

4.1.6 Outcome Measures

The technical outcome measures directly associated with methods were:

1. Acceleration - measured directly from the vibration platform, foot, and body of the thigh muscle.
2. Frequency - derived from acceleration.
3. Displacement - derived from acceleration.
4. Proportion of artefact - derived as a fraction of overall EMG power.
5. Discretised acceleration - time series acceleration data discretised to a single cycle of vibration, averaged and normalised to maximum - Figure 4.5.

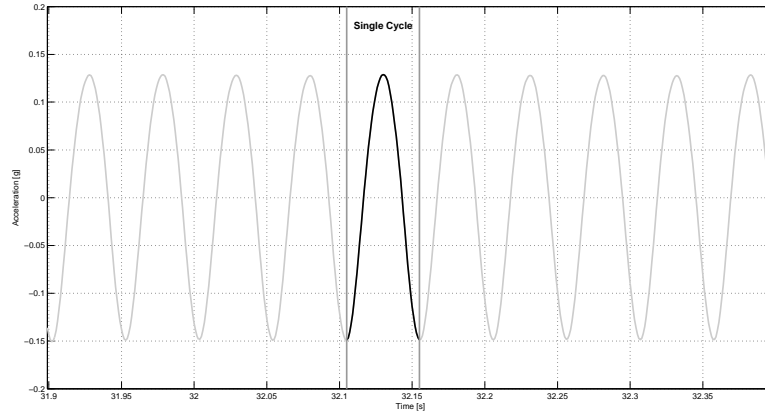


Figure 4.5: Acceleration recorded during a single discrete WBV test presented in the time domain. The troughs of the acceleration signal are used to identify the beginning and end of a single cycle of WBV.

The physiological outcome measures were:

1. EMG - measured directly from the TA, M-GA, L-GA, VL, RF, and BF - Figure 4.6.
2. EMG_N - derived from the measured EMG by calculating the Root Mean Square (RMS) and normalising to the Maximum Voluntary Contraction (MVC).
3. ΔEMG - the relative change from in EMG activity during Vibration when compared to baseline.
4. Discretised EMG - time series EMG data discretised to a single cycle of vibration, averaged and normalised to maximum.

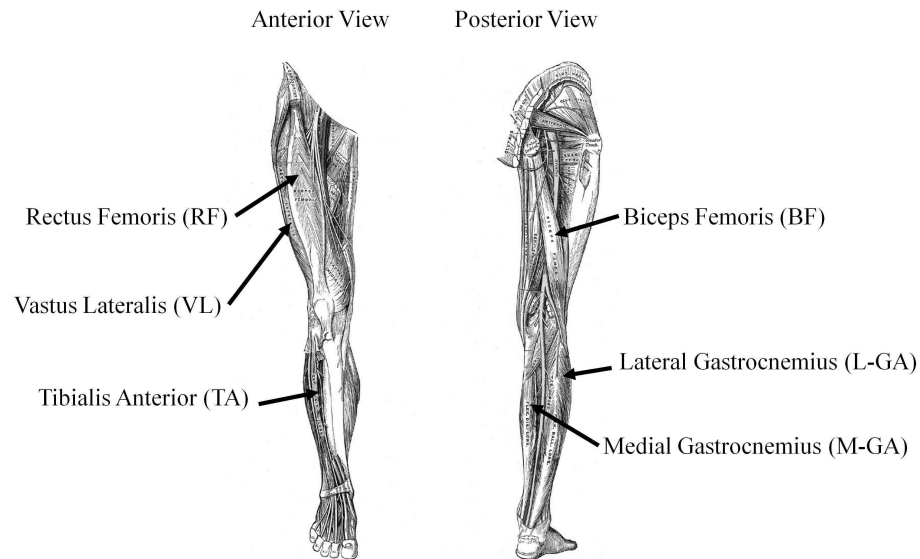


Figure 4.6: Cutaway schematic showing muscle groups for EMG measurements (adapted from Gray [175]).

4.2 Analysis Methods

4.2.1 Summary of Analysis Methods

The off-line filtering and analysis techniques used to report the recorded data are summarised below. Supporting information in the form of figures or worked examples are provided where appropriate.

- **4.2.2: Accelerometer Analysis:** Vibration frequency, average acceleration (RMS) and vibration amplitude were calculated from the raw acceleration data recorded from the vibration platform, foot and thigh.
- **4.2.3: EMG Pre-Processing:** Off-line digital filtering of each EMG was carried out according to current standard guidelines [176].
- **4.2.4: Artefact Quantification:** The magnitude of vibration artefact in every EMG was quantified.
- **4.2.5 EMG: Artefact Removal:** Additional off-line digital filters were applied to each EMG to eliminate vibration artefact.
- **4.2.7: EMG Amplitude Estimation:** The amplitude of the filtered EMG was estimated by computing the RMS of the data.
- **4.2.8: EMG Classification:** Platform acceleration was used to automatically classify the different epochs of the testing protocol: pre-Vibration, Vibration, and post-Vibration. In addition, a custom algorithm was implemented to calculate the Burst-to-Tonic Ratio (BTR) of each EMG. The BTR was used to estimate variability and reject outlying data.

- **4.2.7: EMG Amplitude Analysis:** The magnitude of the neuromuscular response to WBV was estimated by taking the computed RMS of each EMG and averaging over each epoch of the testing protocol. Data were normalised to baseline and MVC, and then grouped according to pBWS and platform acceleration.
- **4.2.10: EMG Vibration Cycle Analysis:** The temporal relationship between neuromuscular activity and WBV was investigated by normalising each EMG to a single cycle of vibration cycle and then averaging over the entire phase of vibration.

4.2.2 Accelerometer Analysis

Acceleration

All raw acceleration data was smoothed using a fourth order, simple moving average filter. Low frequency drift and the gravity offset were removed by high pass filtering using a fourth order Butterworth filter with a cut-off frequency of 1Hz implemented offline in a manner such that there was zero phase lag. The resultant acceleration (a) was found by combining the individual vectors (a_x , a_y , and a_z) of acceleration:

$$a = \sqrt{a_x^2 + a_y^2 + a_z^2} \quad (4.3)$$

Where a is always > 0 ; a_x = Acceleration in x-direction; a_y = Acceleration in y-direction; a_z = Acceleration in z-direction.

The WBV platform acceleration pattern is sinusoidal. Average acceleration was therefore calculated by computing the RMS of the peak resultant acceleration:

$$a_{RMS} = \frac{a_{peak}}{\sqrt{2}} \quad (4.4)$$

Where a_{RMS} = Average acceleration; a_{peak} = Maximum resultant acceleration.

Displacement

Displacement was calculated by integrating the resultant acceleration to obtain velocity, high pass filtering as described previously with a cut-off frequency of 1Hz and integrating velocity to obtain displacement.

Frequency

Figure 4.7 shows sample acceleration data in the time and frequency domains. The frequency of vibration was found by computing the PSD of the acceleration data and identifying the frequency component with the largest overall power contribution as shown. PSD plots were calculated using the periodogram function the Matlab R2010a Statistical Signal Processing toolbox.

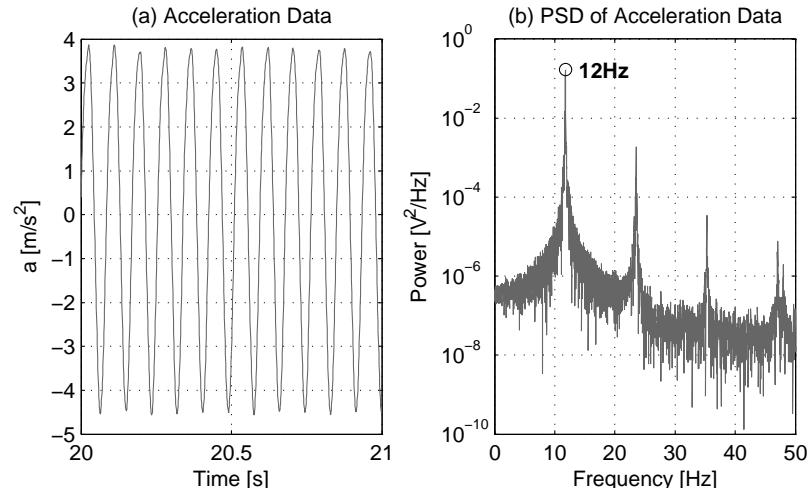


Figure 4.7: Sample acceleration data in the time and frequency domain. The PSD was used to identify the frequency of vibration. PSD = Power spectral density.

4.2.3 EMG Pre-Processing

A standard for recording and reporting high fidelity EMG data was produced by the International Society of Electrophysiology and Kinesiology (ISEK) [176]. These guidelines outline the optimum electrode type, amplification, sampling frequency, filtering and post-processing procedures that should be used. It is recommended that the EMG should be high-pass filtered between 10 - 20Hz, and low-pass filtered between 450 - 500Hz, thus removing low and high frequency noise whilst maintaining the main frequency components of the EMG.

All EMG data was filtered off-line using fourth order butterworth filters in this study. The filters were implemented off-line in a manner such that there was no phase lag. The Bode amplitude plots for the filters used are shown in Figure 4.8. The EMG were high-pass filtered at 30Hz and low-pass filtered at 450Hz. A higher than recommended high-pass filter cut-off frequency was used because considerable high magnitude low frequency noise was found below 27Hz during testing. These high magnitude frequencies were consistent with the frequencies of vibration that were being applied during testing. Line interference was removed using a fourth order, stop-band butterworth filter between 49 - 51Hz.

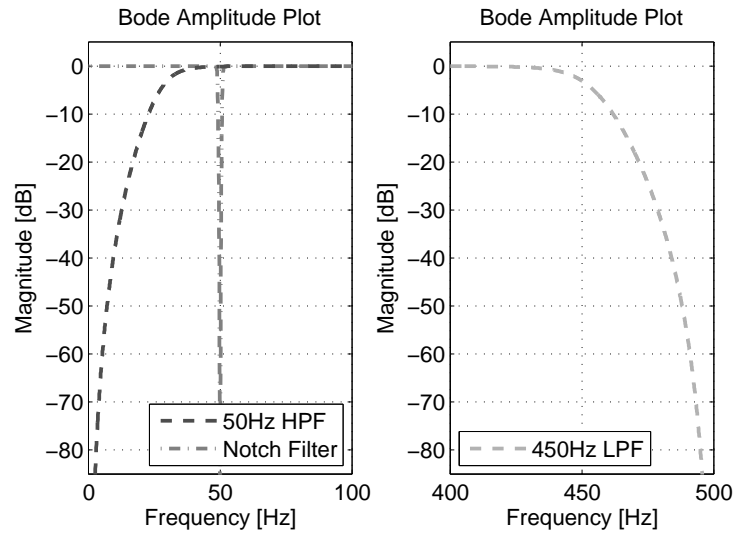


Figure 4.8: Bode amplitude plots for the zero-phase, fourth order, Butterworth filters to process the EMG data off-line.

4.2.4 Artefact Quantification

Artefact Effect

The ISEK guidelines are predicated on appropriate preparation, placement, and attachment of the EMG recording electrodes. Furthermore, the guidelines assume little or no movement of the electrode with respect to the muscle fibers from which they are recording. A change in the shape of the EMG would occur in the event that the recording electrode itself had moved during testing. The change in the shape of the EMG is described as an artefact. An example of an EMG artefact caused by movement of the electrode during WBV is shown in Figure 4.9.

Artefact Example

Figure 4.9 shows a sample of EMG data that was recorded from the M-GA during an acute bout of WBV. The EMG data is presented in the time and frequency domains. The data in the left column was not filtered off-line. The data in the right column was filtered according to the standards outlined in Section 4.2.3.

Clear increases in the power of the EMG were identified at specific frequencies when the data was viewed in the frequency domain. It was noted that these frequencies were harmonics of the frequency of vibration, and that filtering according to the standards outlined above did little to reduce the amplitude of these frequencies in either the time or frequency domain. It was hypothesised that these harmonic frequencies were the result of vibration induced movement of the EMG recording electrode during WBV.

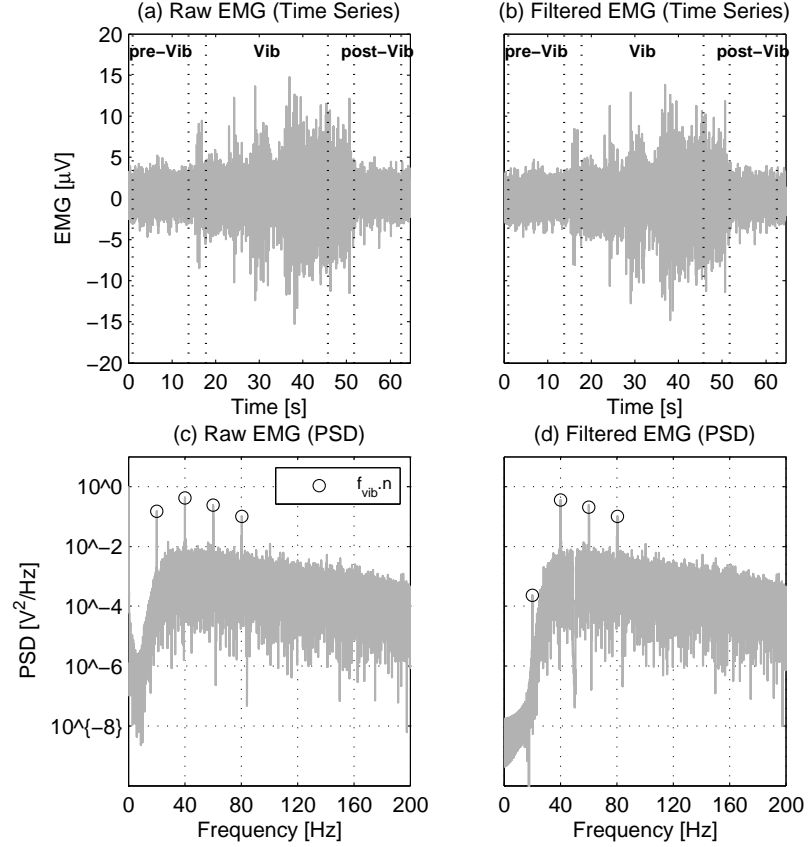


Figure 4.9: Example of raw and filtered EMG data. The data was recorded from the M-GA during an acute bout of WBV at a frequency of vibration of 20Hz. The EMG are presented in the time ((a) & (b)) and frequency ((c) & (d)) domain. Vibration induced movement artefacts are highlighted in the frequency plots. Plots (a) & (b) and plots (c) & (d) have the same y-scale respectively. Note that applying off-line digital filters according the the ISEK standard has a negligible effect on the magnitude of the EMG ((a) vs (b)) and reduces low frequency noise only as seen in (d). PSD = Power Spectral Density; f_{vib} = Frequency of vibration; $f_{vib.n}$ = Integer multiples of frequency of vibration.

Artefact Power

The relative magnitude of each harmonic (p_{h_n}) of vibration was calculated as a fraction of total signal power using Equation (4.5). Total signal power (P_{all}) was found by computing and integrating the PSD across all frequencies. Integer multiples (n) of the frequency of vibration were assumed to be the harmonic frequencies at which artefact occurred (h_n). The total power of each harmonic (P_{h_n}) was then calculated by integrating the PSD within a frequency band of $h_n \pm 1\text{Hz}$.

$$p_{h_n} = \frac{P_{h_n}}{P_{all}} \quad (4.5)$$

Where p_{h_n} = Magnitude of each harmonic; P_{all} = Total signal power; P_{h_n} = Total signal power in specified frequency range.

For the data shown in Figure 4.9, p_h for the first four harmonics was found to be $0.051\text{V}^2/\text{Hz}$ and $0.054\text{V}^2/\text{Hz}$ for the raw and filtered data respectively. This indicates that 5% of the total power of the EMG signal was contained within the first four harmonics of vibration, i.e. within the frequency ranges of 19 - 21, 39 - 41, 59 - 61, and 79 - 81Hz.

The total contribution of all harmonic frequencies (p_h) was calculated by integrating the contribution of each individual harmonic (p_{h_n}) using Equation (4.6):

$$p_h = \sum_{i=1}^n p_{h_n} \quad (4.6)$$

4.2.5 Vibration Artefact Removal

Artefact was removed from the EMG recorded in this study using a fourth order, zero-phase, stop-band ($h_n \pm 1\text{Hz}$) Butterworth filters. Bode amplitude plots are shown in Figure 4.10, representing the stop-band filter for the first harmonic of each frequency of vibration. Validation of this filtering technique is described in Section 5.1.2 using artificially generated EMG data.

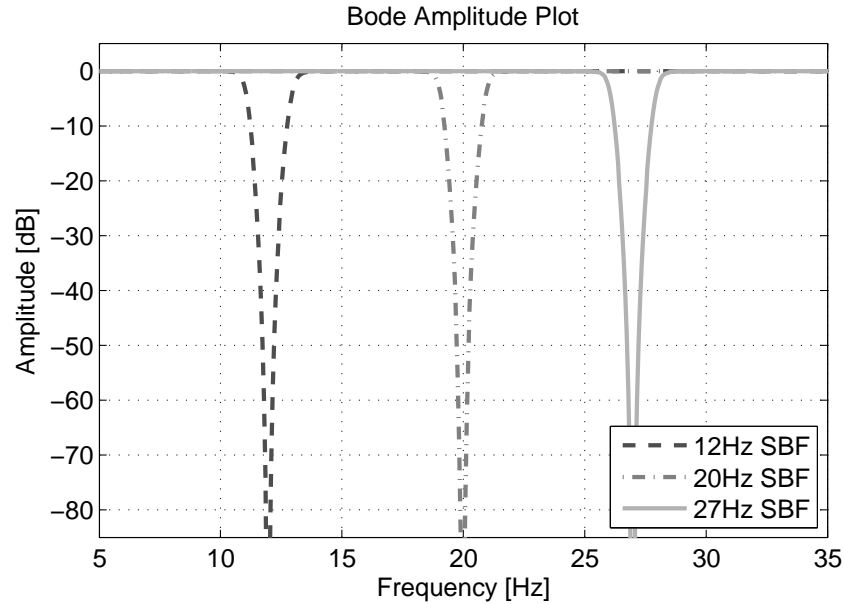


Figure 4.10: Bode plots. SBF: Stop Band Filter.

By applying these stop band filters to the data presented in Figure 4.9, p_h for the first four harmonics was reduced to less than $0.001\text{V}^2/\text{Hz}$.

4.2.6 SCI Case Study

The presence of vibration induced motion artefacts in EMG recorded during WBV was confirmed by carrying out a case investigation of Subject S11_{SCI}. Subject S11_{SCI} suffered a traumatic Lower Motor Neuron (LMN) lesion which principally affected the shank muscles. The WBV-pBWS experimental test configuration allowed the application of the WBV in

a more conventional manner. In this particular instance it was therefore known that a neuromuscular response from the areflexic shank muscles to WBV was impossible, but was possible from the spastic thigh muscles. A randomly selected raw data set (vibration frequency = 20Hz, vibration amplitude = 2mm, pBWS = 50%) was reviewed in the time and frequency domains. PSD plots were reviewed to specifically identify artefact ‘spikes’ in the EMG recorded from the TA and VL. The targeted stop-band filters described above were then applied and the data was reviewed in the time and frequency domains again.

4.2.7 EMG Amplitude Estimation

Every acquired EMG was filtered according to the steps outlined above (Sections 4.2.3 & 4.2.5). The amplitude of the EMG was then calculated by fully rectifying the data and computing the RMS using Equation (4.7):

$$x_{RMS}(t_n) = \sqrt{\frac{\sum_{i=1}^n x(t_{n-i})^2}{n}} \quad (4.7)$$

In this study, the RMS of the entire EMG was calculated using 0.2s (200 samples at 1kHz sample rate) moving, x =EMG data; n =Window size [s]; t =time [s].

4.2.8 EMG Classification

The aim of this study was to measure and quantify the steady-state, acute neuromuscular response before, during and after WBV with pBWS. By definition, the measured response, and therefore contraction, must be predominantly tonic in nature to be considered valid. A tonic contraction was confirmed when the neuromuscular response was uniform, maintained and reproducible for a given set of experimental conditions.

An initial visual qualification of the recorded EMG data was therefore carried out. Random and inconsistent burst-like increases in neuromuscular activity were identified in some instances. These instances were attributed to tripping or spasm and did not meet the criteria set out above. Such instances were readily identified as a burst in activity superimposed on a tonic muscular contraction, and were quantified by calculating the ratio of burst-like to tonic activity. This ratio is described as the Burst-to-Tonic Ratio (BTR) hereafter.

A custom algorithm was used to: (I) split the EMG into either quiescent (pre-vibration or post-vibration) or active (vibration) epochs, (II) calculate the BTR, and (III) reject invalid data sets with a high BTR value. This algorithm is described using a worked example in the following subsections.

(I) Epoch Isolation

Vertical platform acceleration (longitudinal axis, Figure 4.2) was used to identify the three epochs of interest, which were: (i) pre-Vibration (the start of the test to the start of vibration), (ii) Vibration (the start of vibration to the end of vibration), and (iii) post-Vibration (the end of vibration to the end of the test). Transient changes between epochs were eliminated by ignoring the first 2s and last 2s of data from each epoch. The WBV platform was ramping

up and down from the target vibration frequency during these transient periods and was therefore not of interest. Three separate and unique EMG were thus output from this step.

(II) Burst-to-Tonic Ratio

The custom algorithm used to compute the BTR is described using artificially generated sham data (2Hz sinusoid overlaid with uniformly distributed pseudorandom noise). Sham data is used in this instance to simplify the description of the BTR computation algorithm. An example of this same process applied to real EMG data is shown using a worked example at the end of this section. A 0.5s sample of sham data (x) is shown in Figure 4.11(a). This is typical of a fully rectified EMG. Large spikes in amplitude are seen with no discernible underlying level of tonic activity. The RMS of this data was calculated using Equation (4.7) and is shown in Figure 4.11(b). A consistent underlying level of ‘tonic’ activity in addition to some interspersed burst-like activity is now easily identified. Tonic activity was defined as the absolute minimum level of activity, and was identified as the minimum (x_{min}) computed RMS value shown in Figure 4.11(b).

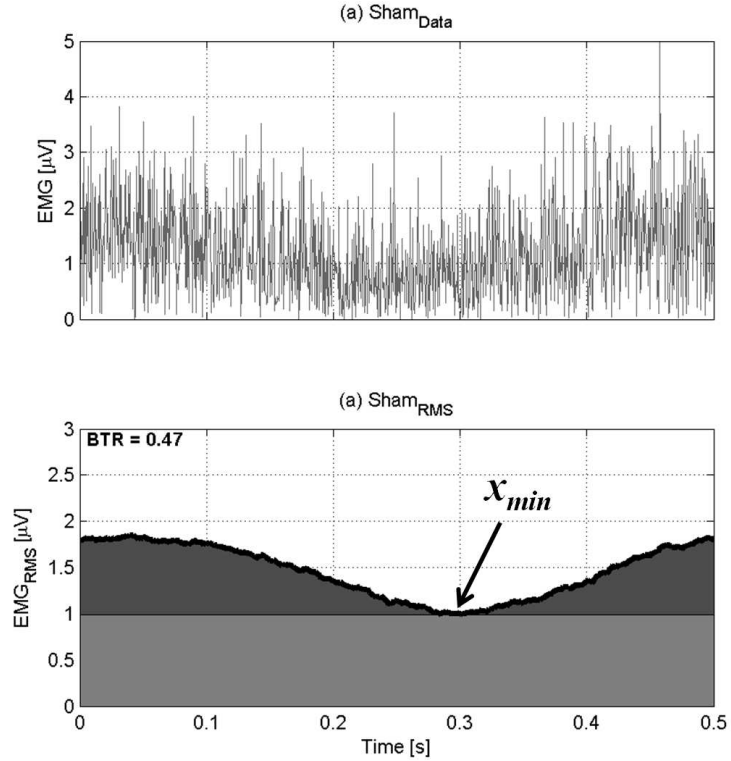


Figure 4.11: Schematic describing process for computing BTR.

Tonic activity (x_{tonic}) was calculated using Equation (4.8) by integrating the minimum level of EMG activity with respect to time:

$$x_{tonic} = \int_{t_0}^{t_1} x_{min}(t) dt \quad (4.8)$$

Where x_{min} = Minimum x_{RMS} as identified in Figure 4.11(b).

Burst activity (x_{burst}) was calculated in a similar manner using Equation (4.9):

$$x_{burst} = \int_{t_0}^{t_1} x_{RMS} - x_{min}(t) dt \quad (4.9)$$

Where x_{RMS} was calculated using Equation (4.7).

The BTR was calculated as the ratio between these two values:

$$BTR = \frac{x_{burst}}{x_{tonic}} \quad (4.10)$$

(III) Outlier Rejection

The magnitude of tonic muscle activity for a given epoch (pre-Vibration, Vibration or post-Vibration) was defined in this analysis as the minimum EMG_{RMS} value for the entire epoch in question. A BTR value was then calculated and assigned to each consecutive 0.5s window of the epoch. The matrix presented in Table 4.5 was used then used to determine if the EMG data for the epoch was either valid or an outlier.

Table 4.5: EMG outlier rejection matrix. EMG = Electromyogram; BTR = Burst-to-Tonic Ratio; *Must be one continuous period to meet the definition of steady-state.

Condition	BTR	Duration	Outcome
1	≤ 5	Entire EMG	Retain entire sample for further analysis
2	≤ 5	6s*	Retain 6s sample only for further analysis and reject remainder as outlier
	≥ 5	Remainder of EMG	
3	≤ 5	<6s	Reject entire EMG as outlier
	≥ 5	Remainder of EMG	

An example of the application of this procedure to real EMG data is described in the following worked example.

Worked Example

Figure 4.12(a) presents an EMG that was recorded from the medial gastrocnemius during a discrete WBV test. The EMG was first filtered off-line, fully rectified, and the RMS calculated using Equation (4.7). The EMG was next discretised in time and split into the relevant epochs of the test: pre-Vibration (2 - 16s), Vibration (19 - 51s), and post-Vibration (55 - 66s). These epochs are shown in Figure 4.12(a). The RMS of the EMG for the Vibration epoch (Figure 4.12(b)) and post-Vibration epoch (Figure 4.12(c)) are shown. The thresholds for burst and tonic activity for each epoch were identified. The threshold for tonic activity was identified as $5.34\mu V$ for the Vibration epoch and $3.41\mu V$ for the post-Vibration epoch.

The BTR was calculated based on these threshold values using Equations (4.8 - 4.10). A BTR value in excess of 5 was found at approximately 22s of the Vibration EMG as seen in Figure 4.12(d). This portion of the EMG was therefore rejected. A BTR value of less than 5 was found for the remainder of the EMG. This portion matched ‘Condition 2’ in Table 4.5 and was retained for further analysis.

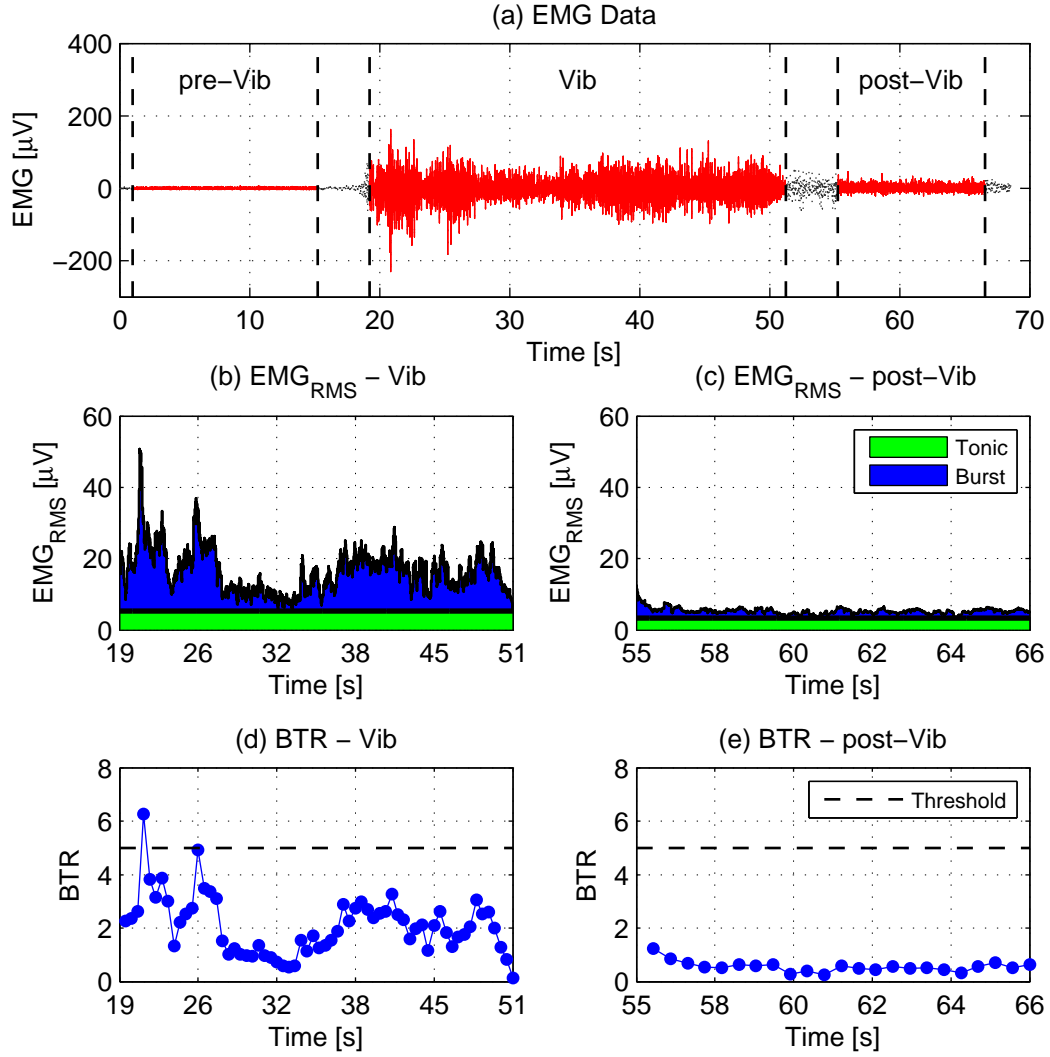


Figure 4.12: Example of BTR calculation using real EMG data. The raw EMG data was acquired from the M-GA during a WBV test at a vibration frequency of 27Hz and vibration amplitude of 3mm with 50% pBWS provided. Standard offline filtering including vibration artefact removal was applied. The filtered EMG is shown in plot (a). The recorded vertical platform acceleration was used to automatically split the EMG into the three different epochs: pre-Vibration, Vibration and post-Vibration. The beginning and end of each of epoch is shown in plot (a). The RMS of each epoch of EMG was calculated. The minimum EMG_{RMS} value for each epoch was identified and taken to be the magnitude of tonic muscle activity for that epoch. Tonic and burst activity are shown in the plots for the Vibration (b) and post-Vibration (c) epochs. The BTR was then calculated for each 0.5s window of each epoch. Each calculated BTR value is shown for the Vibration (d) and post-Vibration (e) epochs. The threshold for rejecting a portion or an entire EMG was set at a BTR value of 5. This threshold is shown for the Vibration (d) and post-Vibration (e) epochs. Portions of the EMG which exceeded this threshold were considered to be outliers.

4.2.9 EMG Amplitude Analysis

Neurologically Intact

In summary, the pre-processing steps carried out on the EMG data were: off-line filtering, full signal rectification, computation of the RMS and outlier rejection. The neuromuscular response was quantified by calculating the mean of the EMG_{RMS} for each test epoch: pre-Vibration (\bar{x}_{prv}), Vibration (\bar{x}_v), and post-Vibration (\bar{x}_{pov}).

Absolute neuromuscular activity (EMG_N) was quantified by normalising to a MVC using Equation (4.11).

$$EMG_N = \frac{\bar{x}}{EMG_{MVC}} \quad (4.11)$$

Where EMG_{MVC} = The maximum RMS value found during the MVC. EMG_N can be any value between 0 and 1. Research has shown that there is a linear relationship between the myoelectric signal, as recorded using EMG, and motor recruitment and force output [177, 178]. A value of 1 thus implies that the muscle is contracting at its maximum capacity. In practice it is not possible to get a value of zero, however as EMG_N tends towards 0, it implies that the magnitude of the contraction is small.

The relative change in neuromuscular activity (ΔEMG) was quantified by referencing \bar{x}_v to baseline using Equation (4.12). Baseline was defined in this analysis as \bar{x}_{prv} .

$$\Delta EMG = \frac{\bar{x}_v - \bar{x}_{prv}}{\bar{x}_v} \quad (4.12)$$

ΔEMG can be any value between -1 and 1. A ΔEMG value equivalent to 1 implies that the neuromuscular response during vibration is much larger than the response before vibration. A value equal to 0 implies that there was no change in neuromuscular activity. A value equivalent to -1 implies that there was an attenuation of the neuromuscular response during vibration.

Throughout the presentation of the results, the test conditions - vibration frequency (f) and vibration amplitude (d) - were related by the theoretical average platform acceleration (a_{theor}) which was calculated using Equation (4.13). This value was used group, compare and contrast the results for different experimental conditions.

$$a_{theor} = \frac{4\pi^2 f^2 d}{\sqrt{2}} \quad (4.13)$$

Where a_{theor} = theoretical average platform acceleration; f = vibration frequency; d = vibration amplitude. According to this relationship, frequency and displacement are related to platform acceleration as described in the table below.

Table 4.6: Relationship between acceleration, frequency, and amplitude

Frequency [Hz]	Amplitude [mm]	Acceleration [g]
0	0	0.00
12	1	0.41
12	2	0.82
20	1	1.14
12	3	1.23
27	1	2.07
20	2	2.28
20	3	3.41
27	2	4.15
27	3	6.22

SCI

The methodology described above was modified slightly for the SCI participants. Normalisation of the EMG to the maximum voluntary capacity of the muscle is dependent on a reliable MVC which was not achieved for many of the SCI participants. Therefore, the EMG was split into the pre-Vibration, Vibration and post-Vibration epochs for the SCI participants and the EMG_{RMS} was calculated. The data was then normalised by calculating ΔEMG which described the relative change in EMG activity to the pre-Vibration epoch. Absolute EMG activity was taken as the average of the EMG_{RMS} recorded during the Vibration epoch only.

4.2.10 EMG Vibration Cycle Analysis

The EMG Amplitude Analysis described above is predicated on the RMS calculation and focussed on the steady-state neuromuscular response to WBV to this point. Steady-state was determined by assessing the variability of the EMG by calculating the BTR. Data sets with a large BTR were deemed as variable and not in a ‘steady-state’ and were therefore excluded as outliers. The data included in the analysis were therefore concluded to be in a steady-state and in an ideal case the EMG would have been ‘flat’ for the duration of the Vibration epoch. The magnitudes of the steady-state EMG were then determined by calculating the RMS of the data.

This analysis methodology implies that the neuromuscular response over a period of 10s, for example, is equivalent in magnitude to the response over a single vibration cycle period. However, it is known that an EMG response is not flat in practice but is rather a combination of multiple CMAP’s which are separated spatially and in time. The EMG is a visual manifestation of the summation of all CMAP. The RMS calculations used a 0.2s moving window and is robust when estimating the amplitude of a static or sustained muscle contractions [179], however in practice some short duration characteristics of the EMG may be lost, for example an instance of high amplitude neuromuscular activity occurring once per vibration cycle. Short duration characteristics may be lost because the RMS calculation is fundamentally an averaging technique which is predicated on the selected window size. Any change in the EMG which may occur within a duration less than the window size, say 1/12 seconds (0.083 seconds), 1/20 seconds (0.05 seconds) or 1/27 seconds (0.037 seconds), would

therefore be averaged out. Therefore, fast acting dynamic changes in neuromuscular activity occurring during a single vibration period would not be detected from an RMS estimation applied to an EMG.

Such characteristic changes have previously been proposed by Ritzmann *et al.* [88], whereby ‘phase-locking’ of the EMG during WBV was shown to occur. Ritzmann *et al.* described how the neuromuscular response is locked in phase with the vibration frequency during phase-locking. They identified a peak in the EMG which occurred once during each single cycle of vibration. The peak was larger in amplitude than the response at all other points in the vibration cycle. In addition, they found that the occurrence of the peak had the same latency as a short latency reflex. It was concluded from this study that the neuromuscular response to WBV was based on the stretch reflex [88].

Therefore, in addition to the *EMG Amplitude Analysis* described above, the neuromuscular response to WBV was investigated by carrying out an additional *EMG Vibration Cycle Analysis*. The pre-processing steps carried for the Vibration Cycle Analysis were: off-line filtering, full signal rectification, and outlier rejection. Each EMG was then discretised for each 360° cycle of the vibration platform, re-sampled such that each period contained the exact same number of data points, and averaged for all cycles. A single 360° cycle of vibration was defined as beginning at the lowest point (trough) of the platform acceleration and ending when the platform next returned to this point. Vertical platform acceleration (longitudinal axis, Figure 4.2) was used to identify each vibration cycle in a data set. The corresponding EMG and acceleration data was then isolated and averaged to represent a single vibration period. In this way, an EMG recorded during a 1s period of 20Hz vibration yielded a total of 20 discretised EMG. The average of these discretised EMG was then taken to smooth the data. It was hypothesised that any unique neuromuscular responses occurring during a single period, should they exist, would become evident while random changes in activity would be minimised by averaging. The response over a single 360° cycle of vibration could therefore be detected.

This analysis was therefore principally qualitative because the shape of the normalised EMG was assessed to determine the relationship between muscle activity and the phase of vibration. This methodology is described hereafter as a Vibration Cycle Analysis and was carried out on every EMG that met the inclusion criteria described in Section 4.2.8.

Data Re-sampling

The exact number of discrete data points per 360° vibration cycle for a given sampling frequency (f_s) and vibration frequency (f_v) is given by Equation (4.14) under ideal circumstances:

$$S = \frac{f_s}{f_v} \quad (4.14)$$

Where S = number of data points; f_s = sampling frequency; f_v = vibration frequency.

Thus, an EMG sampled at a frequency of 1000Hz should yield 50 data points per vibration

period at $f_v=20\text{Hz}$, or 37 data points per vibration period at $f_v=27\text{Hz}$. This is rarely the case with a discrete recording setup in practice. More or fewer data points per vibration cycle than the ideal case above is common. It is therefore necessary to standardise the number of data points in each vibration cycle to allow for the comparison of the results from the different test conditions. This was done here by re-sampling the data using a 1 dimensional linear interpolation function in the Matlab Toolbox (MatlabR2010a, The Mathworks Inc., Massachusetts, USA), which yielded exactly 100 data points per vibration cycle.

Worked Example

A graphical representation of this process is presented in Figure 4.13. A 0.4s sample of EMG and vertical platform acceleration data is shown in the left column. Eight full 360° vibration periods (P1 - P8) were identified. The first period (P1) contained approximately 50 discrete data points which were re-sampled such that the period now contains exactly 100 discrete data points. This process was repeated for periods 2 - 8 (P2 - P8). The average of all eight cycles was calculated and the result is shown in the right column.

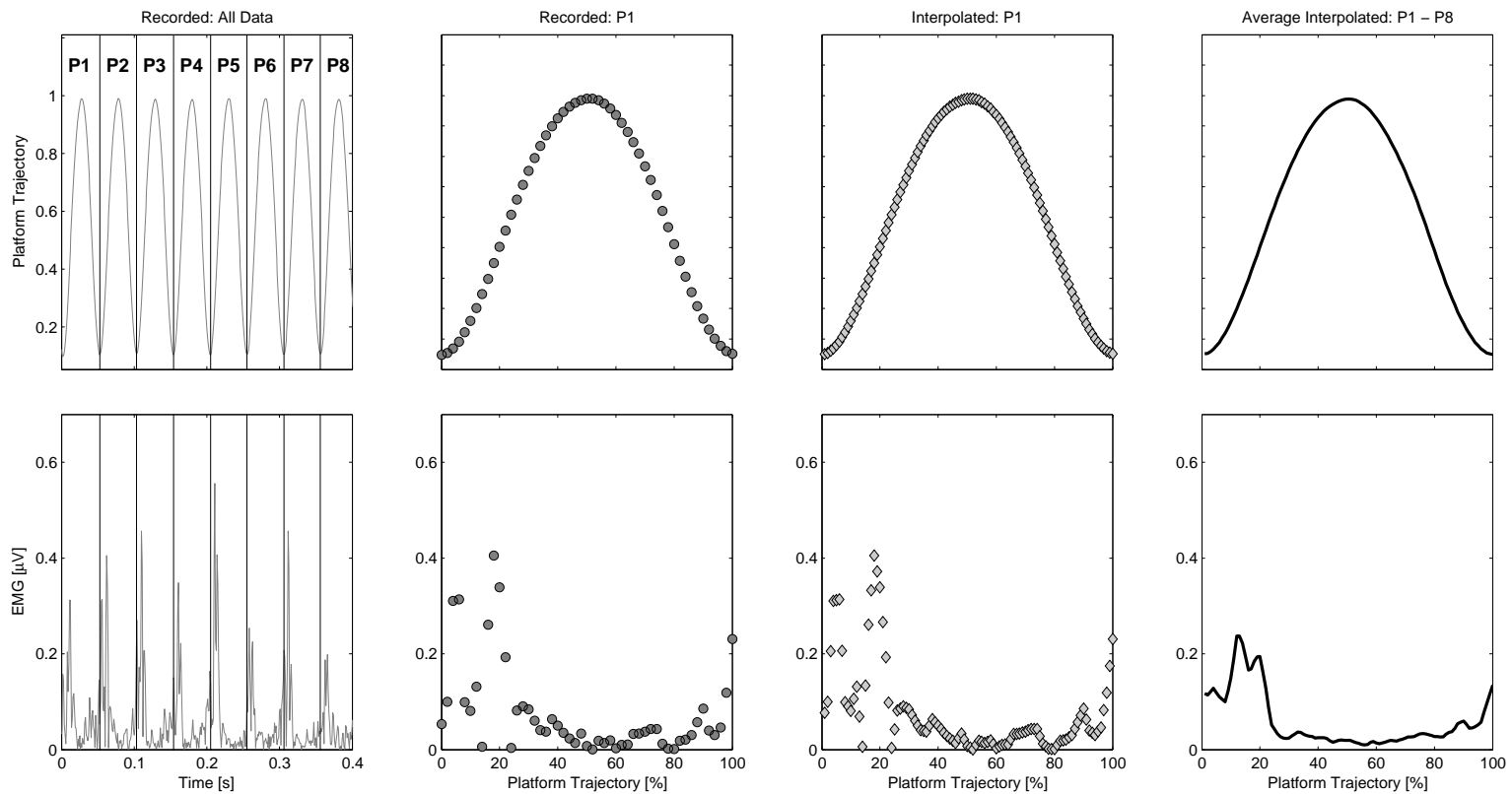


Figure 4.13: Outline of EMG vibration cycle analysis. Real, EMG and acceleration data are presented in the left column. The data were discretised into individual periods (P1 - P8) according to the position of the vibration platform. Each period of data was re-sampled such that it contained exactly 100 data points. The average over all periods was then found and the results are shown in the right column. According to this method, general trends in the EMG response to a single vibration cycle may be observed. Note that platform trajectory was normalised to maximum for presentation in these plots.

4.2.11 Statistical Analysis

This section provides a general overview of the statistical tests used in this study. Additional information about the statistical analyses is provided in the relevant results section where appropriate. An example of such additional information would be the grouping factors used in an ANOVA. All statistical analysis was carried out using the Matlab Statistics Toolbox (MatlabR2010a, The Mathworks Inc., Massachusetts, USA).

Normality

The distribution of grouped data were assessed for normality using the Lilliefors test with a significance detection level of $\alpha \leq 0.05$.

T-test

Paired groups of data were analysed by carrying out a one-sample T-test for paired data or two-sample T-test for independent data. The alternative non-parametric Wilcoxon signed rank and Wilcoxon rank sum tests were each used respectively if the distribution of the the paired groups of data were found to be non-Normal. A significant difference was identified for a p-value of ≤ 0.05 .

Analysis of Variance

Repeated measures data were statistically analysed by carrying out an ANOVA. Grouped data were first assessed for: (i) Normality as described above, and (ii) equal variance (also described as sphericity) by carrying out a Bartlett multiple-sample test for equal variances. In each case, a significant difference was defined for a computed p-value ≤ 0.05 . A multi-way ANOVA was then carried out on the grouped data. In the event that the assumption of sphericity was found to have been violated, a Greenhouse-Geisser correction factor was applied to the computed ANOVA F-statistic and the corresponding p-value was adjusted accordingly.

In the event that an interaction term was found to be non-significant, the ANOVA model was changed to test for main effects only and the relevant statistics were re-calculated. Where statistical significance was identified, a post-hoc multiple comparisons procedure with Bonferroni correction was carried out to identify significantly different pairs of data. The Bonferroni correction was applied to minimise Type II errors [58]. The alternative non-parametric Friedman test was also used in place of the ANOVA where appropriate. Statistical results were tabulated using Table 4.7 as a template:

Table 4.7: Template for tabulating statistical results.

Grouping Factor	Statistical Parameter (DF)	p	GuGI
Factor 1	Parameter 1 (DF)	p-value	Significantly different pairs of data in Factor 1

The following information was noted in the table. Each of these parameters were calculated automatically by the Matlab Statistics Toolbox:

- **Grouping Factor:** The grouping factor under investigation. Vibration frequency, vibration amplitude and type of SCI are examples of different grouping factors which were investigated in this thesis.
- **Statistical Parameter:** The statistical parameter associated with the statistical test being executed. For example, the ‘F’ statistic was calculated for the ANOVA and the ‘ X^2 ’ statistic was calculated for the non-parametric Friedman test.
- **Degrees of Freedom (DF):** Number of degrees of freedom. It was derived from the number of groups within the grouping factor.
- **p:** P-value calculated for the statistical test.
- **Gu_{Gl}:** When significance was indicated by the statistical test a pairwise multiple comparisons procedure was carried out on the data contained within the grouping factor and the results were tabulated in this field. For example, three different vibration frequencies were investigated: 12, 20 and 27Hz. During the pairwise multiple comparison procedure the data recorded at 12Hz would be compared with the data at 20 and 27Hz. Similarly the data recorded at 20Hz would be compared with the data recorded at 27Hz. A † symbol was recorded in this field in the event that all groups of paired data were found to be significantly different to one another. The field was left blank in the event that no significant difference was found between any group of paired data. The pairs which were significantly different were noted in all other cases. The paired data with a statistically significant difference were noted as an upper group (Gu) and lower group (Gl) (i.e. 27Hz_{12Hz} was used to indicate that the data recorded at 27Hz was significantly different to the data recorded at 12Hz).

Grouped data was plotted using the boxplot function in the Matlab Statistics Toolbox throughout this thesis. The box plots show the median, interquartile range, upper extreme value and lower extreme value by default for each set of grouped data. An annotated example is shown in Figure 4.14. Default whisker lengths were used in all cases and were calculated automatically using the standard equations in the Matlab Statistics Toolbox (Equation (4.15) and Equation (4.16)).

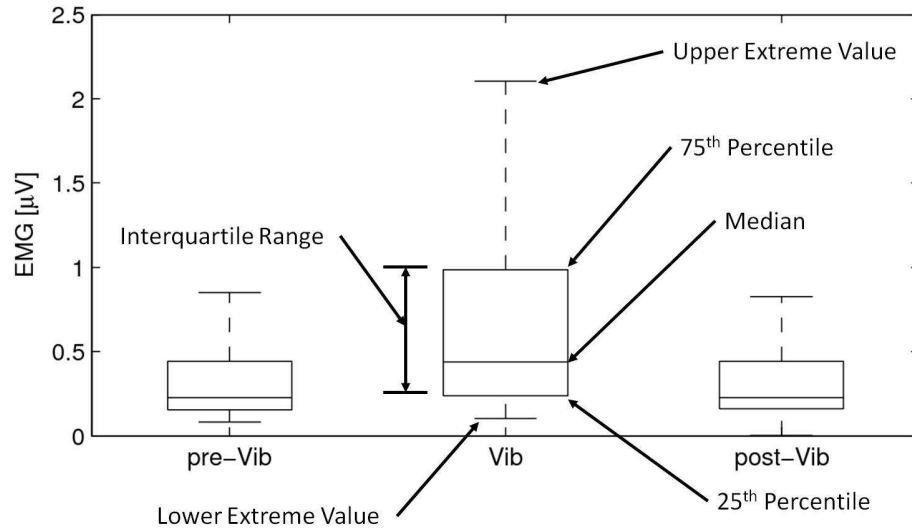


Figure 4.14: Sample box plot.

$$UpperExtremeValue = q_3 + 1.5(q_3 - q_1) \quad (4.15)$$

$$LowerExtremeValue = q_1 + 1.5(q_3 - q_1) \quad (4.16)$$

Where $q_1 = 25^{th}$ percentile and $q_3 = 75^{th}$ percentile. The default value of 1.5 corresponds to approximately $\pm 2.7\sigma$ and 99.3% coverage of normally distributed data.

Effect Size

Effect Size (ES) calculations were based on the Cohen's d statistic, using the pooled standard deviation for the two populations under investigation:

$$\frac{\bar{x}_1 - \bar{x}_2}{\sigma_{1,2}} \quad (4.17)$$

Where \bar{x}_1 = Mean of population 1; \bar{x}_2 = Mean of population 2; $\sigma_{1,2}$ = Pooled standard deviation of population 1 and 2.

A computed ES of 0.1, 0.3, and 0.5 were defined as small, medium, and large respectively [58].

Correlation

Pearson's correlation coefficient was used to assess the pairwise correlation between two or more groups of data.

Chapter 5

WBV Neurologically Intact

A pBWS system was integrated with a WBV platform for this study in order to allow those with a SCI to stand on the vibration platform enabling them to use it in a conventional manner. pBWS also provided a degree of flexibility not provided for by other systems, by allowing the level of support to be adjusted depending on the functional capability of the participant in question. Furthermore, since WBV can be applied in a more conventional manner, this setup allows for a more direct comparison of new findings from SCI participants with existing findings reported in literature. This system was tested with a general population in the first instance in order determine the feasibility of using WBV at reduced loads, and to establish a normative baseline against which the SCI participants could be compared. Furthermore, it provided expertise for the development of an experimental protocol for SCI testing.

Ten participants with no neurological deficit were recruited from the University of Glasgow to participate in two experimental sessions as described in Section 4.1.2. Each experimental session was identical and comprised of 27 discrete test conditions. The vibration was well tolerated and there were no adverse reactions or instances of erythema as described in the literature. The response to WBV was measured using two different EMG amplifiers. Pre-amplification of the EMG at the muscle resulted in a superior quality EMG which was less susceptible to vibration artefact. Despite the improved performance of pre-amplified EMG electrodes versus passive EMG electrodes, a proportion of vibration artefact remained in the EMG and an offline EMG signal processing and classification protocol was required. This protocol was optimized and used for both the general and SCI studies.

5.1 Results

A summary of the results described in this section is presented below for quick reference:

- **Section 5.1.1 Accelerometer Analysis:** Average acceleration, displacement and vibration frequency were calculated for the vibration platform, foot, and thigh. These data indicate the frequency of vibration artefact expected to be present in the EMG recorded during WBV and the magnitude of damping that may be occurring.
- **Section 5.1.2 Artefact Quantification:** The total proportion of vibration artefact

present in the recorded EMG was quantified. Bespoke off-line filters were used to remove the artefact from the EMG.

- **Section 5.1.3 EMG Classification:** The EMG were discretised into pre-Vibration, Vibration, and post-Vibration epochs and outlying data was rejected based on the BTR.
- **Section 5.1.4 General EMG Amplitude Response:** The magnitude of the EMG was quantified by computing and averaging the RMS of each epoch of data. The averaged values were then grouped according to test number, epoch, vibration frequency, vibration amplitude, muscle group and level of pBWS.
- **Section 5.1.5 Specific EMG Amplitude Response:** The averaged EMG_{RMS} data were grouped according to muscle group, vibration frequency, vibration amplitude and level of pBWS.
- **Section 5.1.6 EMG Vibration Cycle Analysis:** The EMG response over a single cycle of vibration was investigated.

5.1.1 Accelerometer Analysis

The root mean square of acceleration, described hereafter as average acceleration, recorded from the vibration platform, foot and VL EMG sensor was calculated for the Vibration period of every test, session and subject using Equation (4.4). The results were grouped according to the theoretical platform acceleration (a_{theor} , Equation (4.13)) and are presented in the boxplots shown in Figure 5.1. The vibration frequency and vibration amplitude parameters related to a_{theor} may be interpreted using Table 4.6.

A review of Figure 5.1 indicated that there was a good correlation between the measured average acceleration and a_{theor} . The correlation between average acceleration of the foot and a_{theor} was poorer, indicating that damping has occurred, resulting in a reduced transmission of vibration to the foot. The average acceleration of the VL EMG sensor was small and unrelated to the level of pBWS, vibration frequency or vibration amplitude.

The measured vibration frequency was determined by computing the PSD of the raw acceleration data and identifying the frequency component with the largest overall power contribution. The frequency of vibration of the vibration platform, foot and VL sensor are shown in Figure 5.2. The data were grouped according to the vibration frequency set for the test and the level of pBWS provided. A visual inspection of Figure 5.2 indicated that there was a strong correlation between the measured frequency and actual frequency for all test conditions, and that the transmission of vibration frequency was unaffected by vibration amplitude or level of pBWS.

Displacement of the WBV platform, foot and VL sensor was calculated by double integration of the recorded acceleration data. The results were grouped according to the vibration amplitude set for the test and the level of pBWS provided. The results are presented as boxplots and are shown in Figure 5.3. A visual inspection of the plots indicated that there was a strong correlation between the measured vibration amplitude and that which was set for the test. Conversely, displacement amplitude of the foot and VL sensor was poorly correlated

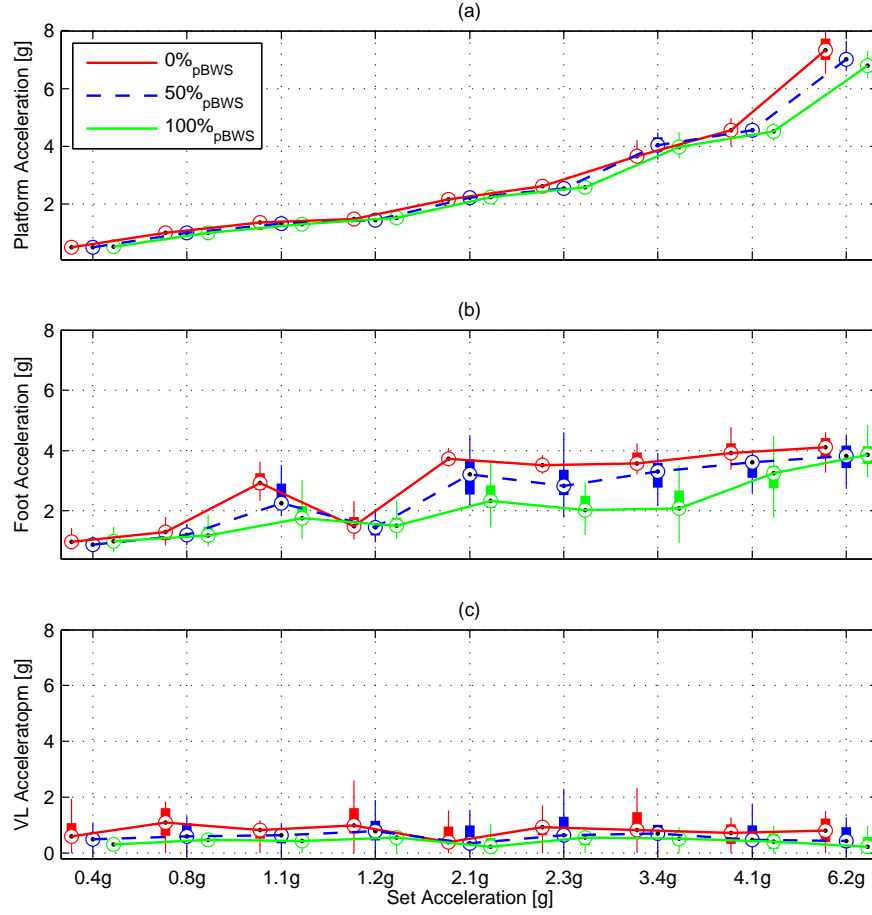


Figure 5.1: Average acceleration data recorded during WBV (Epoch 2 from Figure 4.4 only) from the platform (top panel), foot (middle panel) and VL (bottom panel). The data are grouped according to the expected platform acceleration (a_{theor}) as calculated using Equation (4.13) for the given discrete WBV test conditions, and the level of pBWS provided. The discrete vibration conditions used are identifiable by cross-referencing the a_{theor} values presented on the x-axis above with Table 4.6. The average (median) of each set of grouped data are also shown. Section 4.2.11 provides a detailed explanation of each of the parameters shown in the boxplot above, including: median, percentiles, interquartile range and whiskers.

with that which was set for the test. Furthermore it was unaffected by the level of pBWS provided and inversely correlated with vibration frequency.

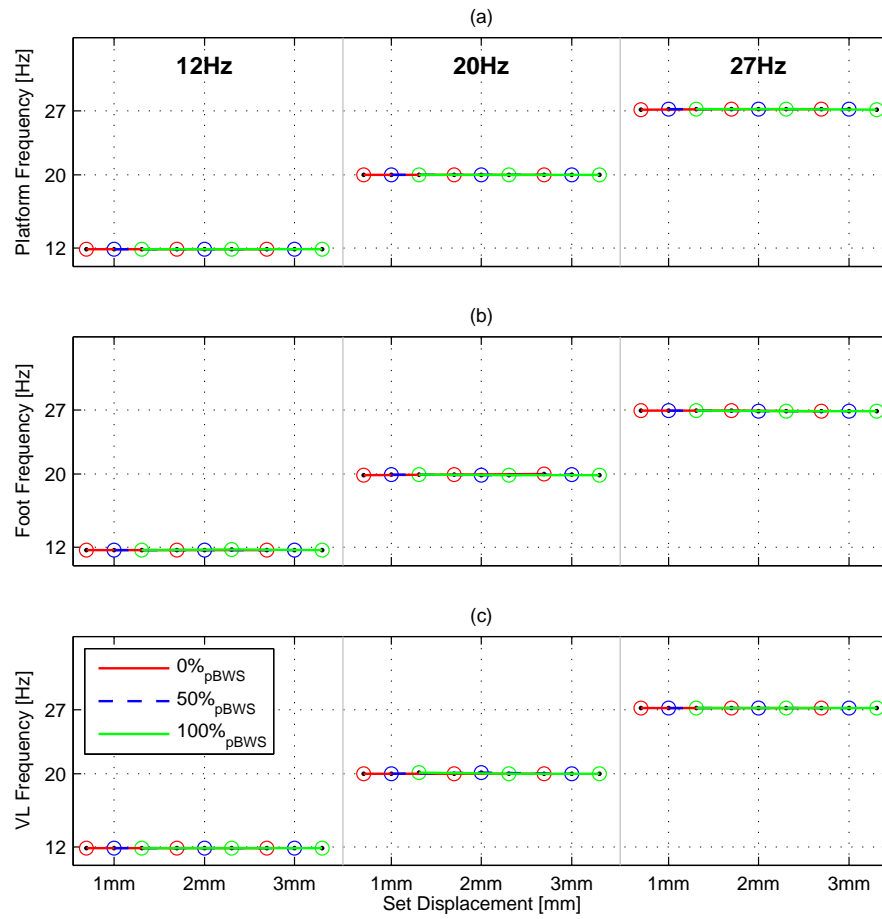


Figure 5.2: Frequency data recorded during WBV (Epoch 2 from Figure 4.4 only) from the platform (top panel), foot (middle panel) and VL (bottom panel). The data are grouped according to the frequency and amplitude of vibration set for the test, and the level of pBWS provided. The average (median) of each set of grouped data are also shown.

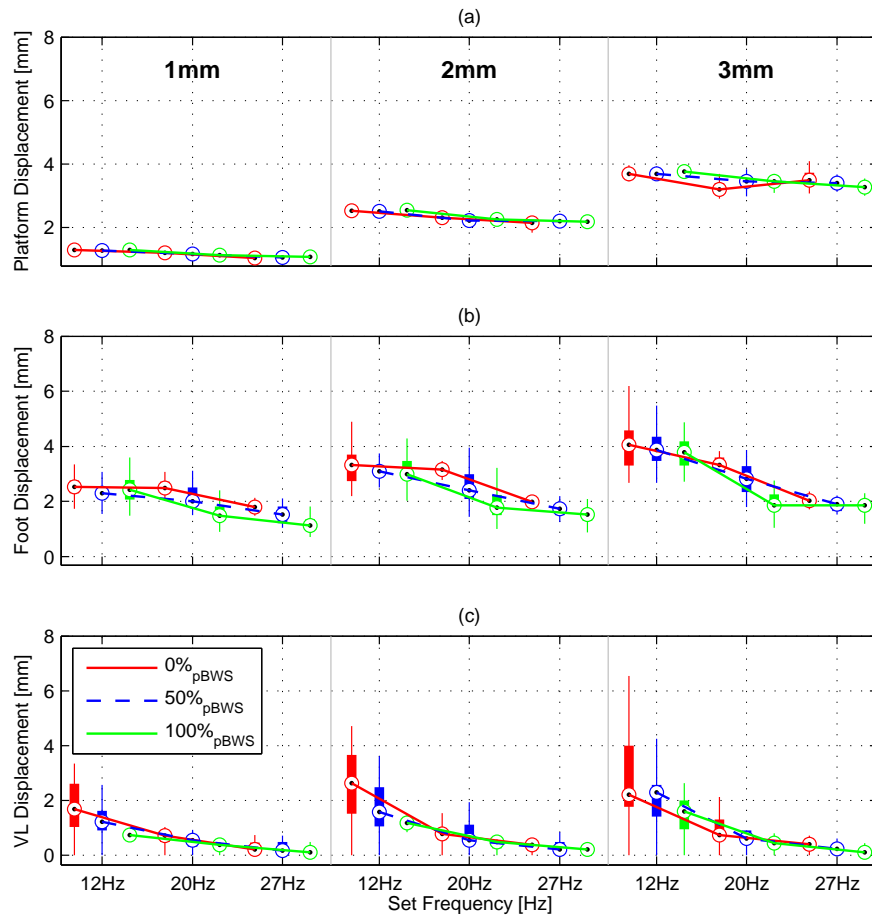


Figure 5.3: Estimated displacement data recorded during WBV (Epoch 2 from Figure 4.4 only) from the platform (top panel), foot (middle panel) and VL sensor (bottom panel). The data are grouped according to the frequency and amplitude of vibration set for the test, and the level of pBWS provided. The average (median) of each set of grouped data are also shown.

Platform acceleration in this application is sinusoidal in nature. Displacement is therefore 180° out of phase with acceleration. The high accuracy acceleration measurements were therefore used to determine the relative movement of the WBV platform, foot and VL sensor with respect to one another. Figure 5.4 shows this relative movement. Acceleration was discretised according to each cycle of vibration, averaged across all cycles and then normalised to maximum thus yielding a movement pattern for each location. These movement patterns are described as trajectories hereafter. The longitudinal component of acceleration is plotted only and the anterior-posterior and medial-lateral components are shown in the appendix. The longitudinal component is the only component shown here because movement during vibration primarily occurred in this direction. The trajectory of the WBV platform was symmetrical and unaffected by pBWS, vibration frequency or vibration amplitude.

Foot trajectory is approximately 180° out of phase with the platform trajectory along the anterior-posterior axis, but almost completely in phase in the longitudinal direction. Foot and VL trajectories similarly demonstrated an inability to maintain synchrony with the vibration platform when the frequency of vibration was increased. This was particularly evident in the VL, possibly indicating a mechanical delay in transmission.

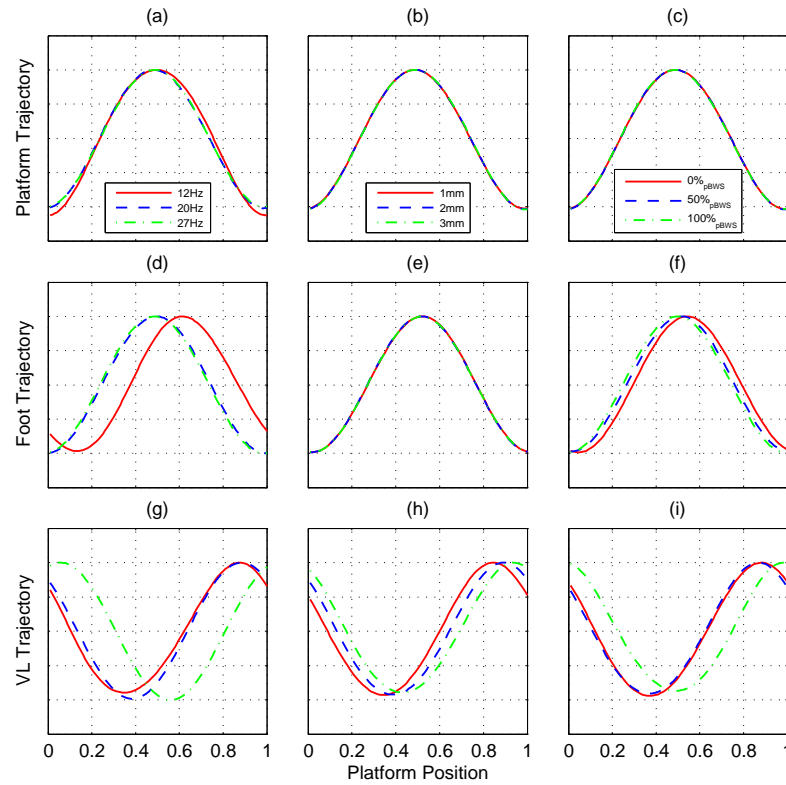


Figure 5.4: Platform ((a), (b) & (c)), foot ((d), (e) & (f)), and VL ((g), (h) & (i)) trajectory were estimated using longitudinal acceleration data recorded from each of these locations during vibration. The data from each discrete test were averaged for each cycle of vibration and normalised to maximum. The y-scale is therefore unitless. The data presented in the left column ((a), (d) & (g)) were grouped according to the frequency of vibration and averaged for all subjects, tests, displacements and levels of pBWS. The data presented in the middle column ((b), (e) & (h)) were grouped according to vibration displacement and averaged for all subjects, tests, frequencies and levels of pBWS. The data presented in the right column ((c), (f) & (i)) were grouped according to the level of pBWS and averaged for all subjects, tests, frequencies and displacements.

5.1.2 Artefact Quantification

This section presents the results of the investigation into the artefact content of the EMG recorded for this study. The analysis focussed on quantifying the vibration induced motion artefact in the EMG. Vibration artefact was only expected to be present in those EMG recorded during vibration. However, in this analysis the entire EMG was analysed (pre-Vibration, Vibration & post-Vibration). This was done to establish a normative data set with which to compare the Vibration EMG and to confirm that the ‘artefact’ frequencies were a result of vibration only and not due to some other unexpected source. This analysis is important because artefact in an EMG could potentially lead to an overestimation of the magnitude of the EMG and therefore an overestimation of the magnitude of the neuromuscular response being attributed to WBV in subsequent analyses. In addition, the optimum EMG recording setup was determined by using two different types of EMG recording electrode and analysing the relative susceptibility of these electrodes to vibration artefact.

Artefact was analysed by first quantifying the power of each harmonic frequency contained within the EMG, and then comparing the relative power of each harmonic with respect to all others. A statistical analysis was then carried out using an ANOVA and Effect Size calculations.

Artefact Power

Every acquired EMG, from all participants and test conditions, was initially filtered according to the filtering steps outlined in Section 4.2.3, and split into the representative epoch: pre-Vibration, Vibration and post-Vibration. The amplitudes of the first 15 harmonics (h_{1-15}) of vibration were then calculated as a proportion of the total power (p_{h_n}) using Equation (4.5). Each value of p_{h_n} (presented as a percentage value of total EMG power) was grouped according to harmonic number (n) and electrode type and the full data set was plotted. Recall that a p_{h_n} value of 0 indicates that none of the power of the EMG is contained within this frequency band, and a p_{h_n} value of 1 indicates that all of the power within the EMG is contained within this frequency band.

Data acquired using the active and passive EMG electrodes were plotted in Figures 5.5(a) and 5.5(b) respectively. Group averages (mean) for each harmonic/electrode combination were also calculated and overlaid the same plots. These group averages were then used to compare the relative performance of the active and passive electrodes and to establish general trends in the occurrence of vibration artefact. Note that the EMG amplifiers were configured with a high-pass filter cut-off frequency of 30Hz, a significant reduction in EMG and artefact amplitude below this frequency was therefore expected. Attenuation of each of the following frequencies was thus observed: $h_1=12\text{Hz}$ & $h_2=24\text{Hz}$ ($f_{vib}=12\text{Hz}$); $h_1=20\text{Hz}$ ($f_{vib}=20\text{Hz}$); and $h_1=27\text{Hz}$ ($f_{vib}=27\text{Hz}$).

No Vibration

The average contribution of any single component of vibration artefact (p_{h_n}) did not exceed 1% during the pre- and post-Vibration epochs, regardless of the type of electrode used or harmonic number. Furthermore, there was typically no difference in the results from either epoch. Maximum values of 6 and 14% were found for the active and passive electrodes respectively.

Vibration - Active Electrodes

The average contribution increased to between 1 and 11% for the active electrodes during vibration depending on harmonic number. A maximum value 80% was found which occurred at the second harmonic. It was noted that the second harmonic occurred at 24, 40 or 54Hz depending on the vibration condition applied. These frequencies were close to or above the high pass filter cut-off frequency used in this study. There was a downward trend in the proportion of artefact at all subsequent harmonic frequencies thereafter.

Vibration - Passive Electrodes

The average contribution of vibration artefact to the total power of the EMG was between 1 and 12% for the passive electrodes during vibration depending on harmonic number. A

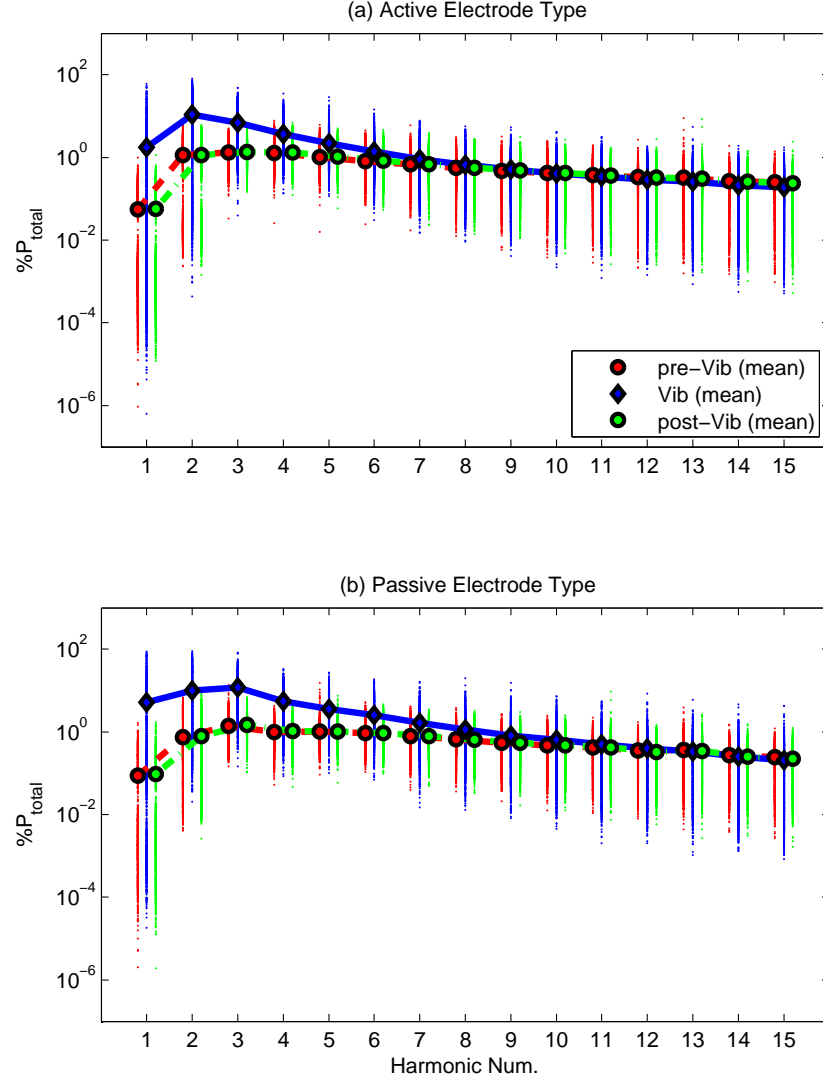


Figure 5.5: The power of the first 15 harmonics of vibration $\pm 1\text{Hz}$ are presented as a percentage of total EMG power (Equation (4.5)), for all subjects, experimental sessions and test conditions. The data acquired using active (a) and passive (b) electrodes are shown separately. Note that the y-scale on each plot is logarithmic and each individual dot is a p_{h_n} value. The data are grouped according to the harmonic number and the epoch of the test condition: pre-Vibration, Vibration and post-Vibration. Averages (mean) are overlaid the grouped data.

maximum values of 90% was found in this instance, which occurred at the third harmonic. There was again a general downward trend in the proportion of artefact at each subsequent harmonic frequency.

The differences between p_{h_n} in the EMG from the Vibration epoch and the pre- and post-Vibration epochs were negligible from the 7th harmonic onwards, regardless of the type of

EMG electrode used. This qualitative observation was quantified with a statistical analysis in a later section. Furthermore, it was concluded that artefact was present in the EMG recorded during the Vibration epoch only. The total artefact content during the Vibration epoch was therefore quantified next. This was done by integrating all values of p_{h_n} (Equation (4.6)) and again grouping according to harmonic number for the active and passive electrodes respectively. Group averages (mean) were calculated and overlaid the same plots. The results for the active and passive electrodes are shown in Figures 5.6(a) and 5.6(b) respectively.

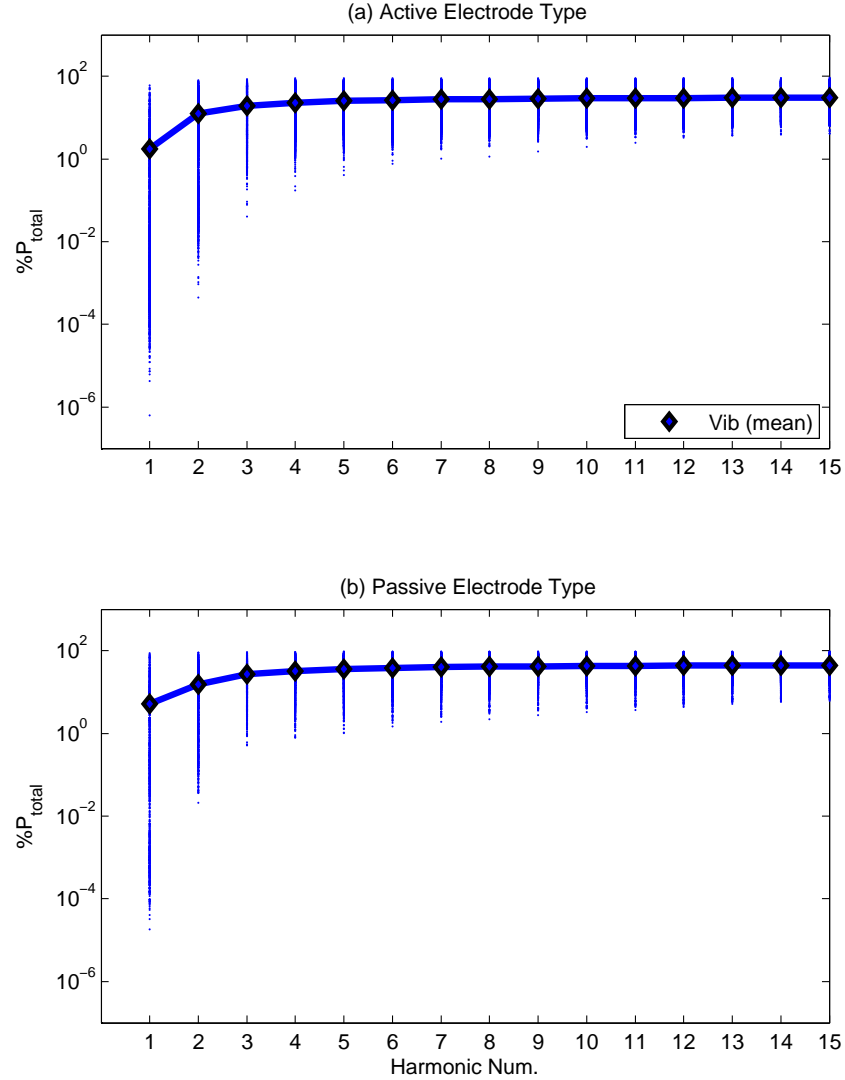


Figure 5.6: The integrated contribution of each individual harmonic p_{h_n} is presented as a percentage of total EMG power (Equation (4.6)), for all subjects and experimental sessions. Each data point is shown as a dot. The data are for the vibration epoch only and are grouped according to harmonic number. The results for the active (a) and passive (b) electrodes are shown separately. Note that the y-scale of each plot is logarithmic. Group averages (mean) are overlaid the same plots.

No Vibration

The total power of the EMG contained within the range of the harmonic frequencies was 10 and 15% for the passive and active electrodes respectively (data not shown on plot) during the pre- and post-Vibration epochs. It was concluded that these values were representative of the coincidental neuromuscular activity that occurred within these frequency bands rather than that from an artificial source.

Vibration

Average values of 23% and 42% were found for the active and passive electrodes respectively during the vibration epoch. Maximal values of 95% were found for certain conditions. These conditions were dependent on the vibration frequency, vibration amplitude, muscle group, level of pBWS and type of EMG electrode.

It is clear from these data that vibration artefact causes a significant increase in the power of the EMG recorded during the vibration epoch. A visual inspection of the data indicates that the majority of the increase occurs between the 1st and 7th or 8th harmonic, with little or no observable change thereafter. A statistical analysis was carried out next to validate this observation.

Statistical Analysis

Statistically significant differences between the groups of data presented in Figure 5.6 were investigated by carrying out a multi-way ANOVA, where harmonic number and test condition (pre-Vibration, Vibration, post-Vibration) were used as the independent grouping factors. The results for the vibration data only are presented in Figure 5.7. A post-hoc multiple comparisons procedure with Bonferroni correction was used to identify significantly different pairs. For simplicity, only those groups of data that were significantly different to the 15th harmonic (h_{15}) are presented in Figure 5.7. The overall ES of these changes was estimated by computing Cohen's d statistic for each consecutive pair of grouped data (Equation (4.17)). Each point presented in Figure 5.7 is the computed ES between h_n and h_{n-1} . An $ES \leq 0.1$ was defined as the threshold value for a small change.

It was found that h_{1-10} (harmonics 1 - 10) caused a significant increase in $\%P_{total}$ when using active or passive EMG electrodes. However, while statistically significant, the overall effect from h_5 and h_6 onwards was deemed to be small for the active and passive electrodes respectively. It was therefore concluded that vibration artefact largely occurred within h_{1-6} only and that it was adequate to remove the up to the 7th harmonic from the EMG. A summary of the typical contribution of these artefacts on the EMG recorded during WBV is described in further detail below.

Artefact Summary

Harmonics h_{1-7} were grouped according to muscle group, vibration frequency, vibration amplitude and level of pBWS to describe the general susceptibility of an EMG to movement artefact under specific experimental conditions. The results for the active and passive electrodes are plotted separately in Figure 5.8. Statistical significance was assessed using

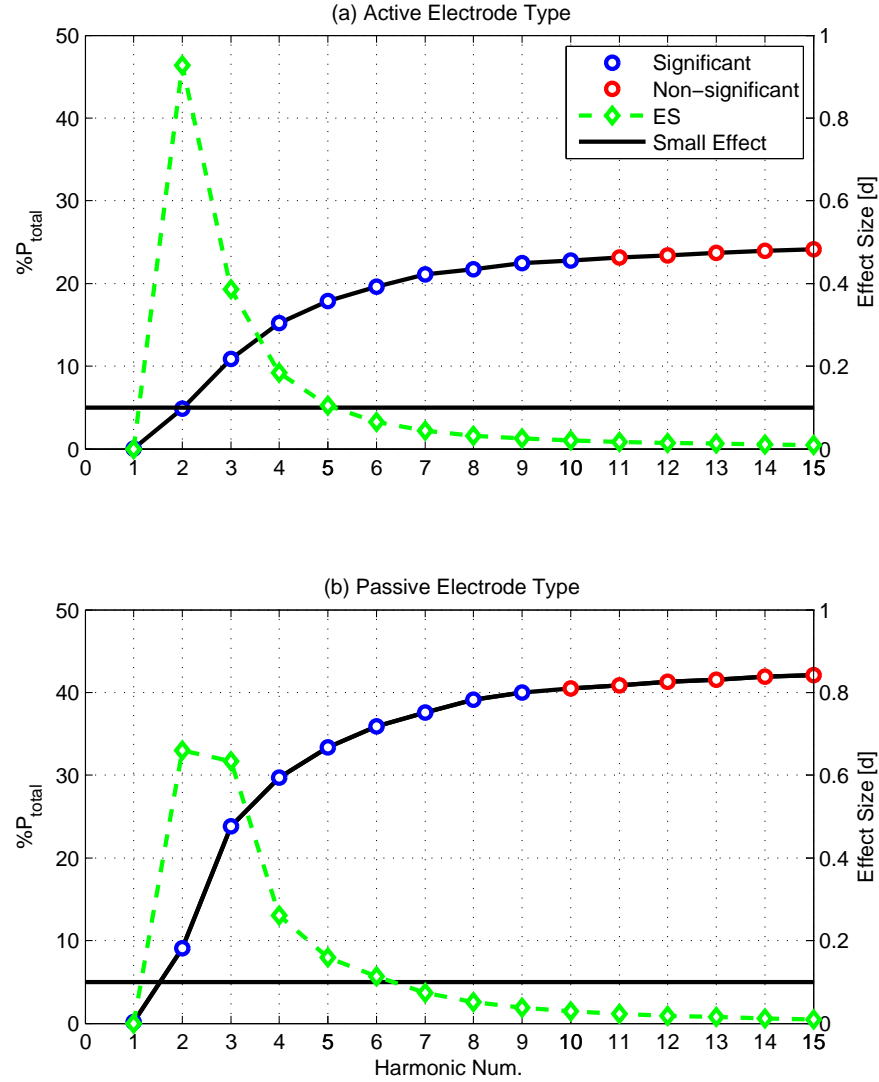


Figure 5.7: The average integrated contribution of the individual harmonics p_{h_n} are plotted with a solid line on the primary y-axis. The values are presented as a percentage of total EMG power recorded during the vibration epoch for all subjects and experimental sessions. The results for the active (a) and passive (b) electrodes are shown separately. Note that these data are the same as those shown in Figure 5.6 and that the y-scale is linear. The corresponding effect sizes are plotted with the dashed line on the secondary y-axis. Effect size was estimated using Cohen's d statistic (Equation (4.17)). The threshold value for a small effect size of 0.1 is also shown on the secondary y-axis. Data groups that were significantly different to h_{15} using an n-way ANOVA and a post-hoc multiple comparisons procedure are highlighted (1 - 10 for active electrodes and 1 - 9 for passive electrodes). ES: Effect size; ANOVA: Analysis of Variance.

a repeated-measures ANOVA, where vibration frequency, vibration amplitude, level of pBWS, and muscle group were treated as the independent factors (Table 5.1). Significantly different pairs were identified using a post-hoc multiple comparisons procedure with a

Bonferroni correction. The EMG recorded using passive electrodes typically contained a larger proportion of artefact than those recorded using active electrodes under the same experimental conditions. It was therefore concluded that only active EMG electrodes should be used during the application of WBV.

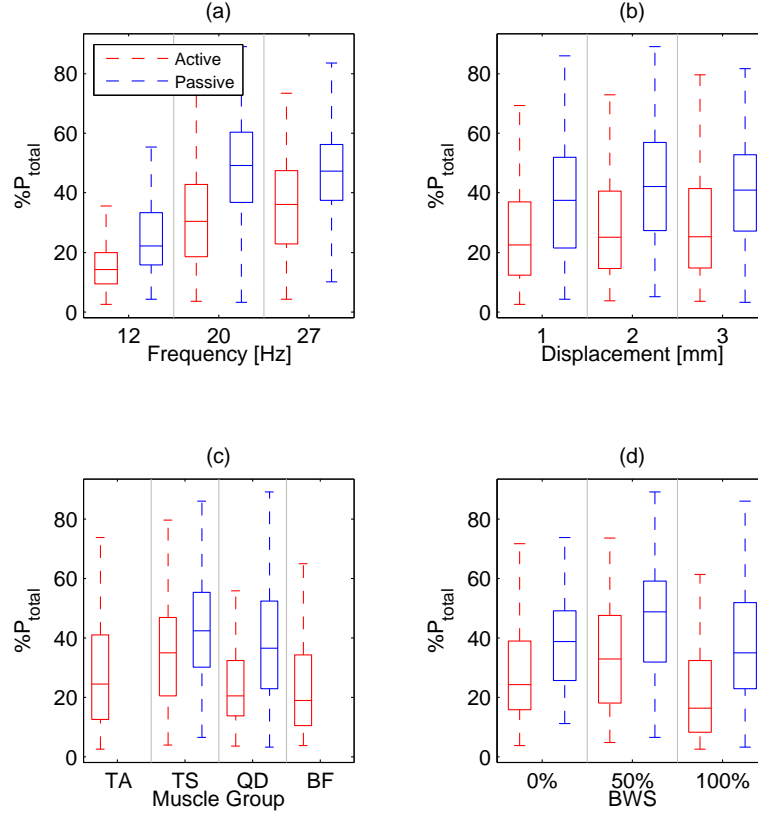


Figure 5.8: General summary of the overall contribution of the first 7 harmonics of vibration to the total power of the EMG. The contribution of the first seven harmonics of vibration were integrated for all subjects and experimental conditions (vibration epoch only) and grouped according to vibration frequency (a), vibration amplitude (b), muscle group (c) and level of pBWS (d). The results from the active and passive electrodes are shown separately. TA = Tibialis Anterior; TS = Triceps Surae muscle group (including Medial Gastrocnemius and Lateral Gastrocnemius); QD = Quadriceps muscle group (including Vastus Lateralis and Rectus Femoris); BF = Biceps Femoris.

A number of additional trends were observed: (i) there was a significant increase in the proportion of artefact in the EMG with vibration frequency, though this trend was less marked for the passive electrodes; (ii) there was a significant increase in the proportion of artefact in the EMG with vibration amplitude; (iii) the presence of artefact was largest in those EMG recorded with 50% pBWS, and smallest in those recorded with 100% pBWS; (iv) there was significantly more artefact in those EMG recorded from muscles which were in closer proximity to the vibration platform (TA, M-GA & L-GA), when compared to those more proximal (VL, BF & RF); and (v) there was no difference in the overall proportion of

artefact in the thigh muscles (VL and BF). The results of the statistical analysis are shown in Table 5.1. These trends were consistent, regardless of the type EMG electrode used. It was therefore concluded that h_{1-7} should be removed from EMG recorded during WBV using appropriate stop-band filters, analogous to those used to remove line interference.

Table 5.1: Statistical results from an analysis of the combined contribution of the first 7 harmonics of vibration artefact (h_1 to h_7) in EMG recorded during WBV using active and passive electrodes.

Parameter	Active Electrode		Passive Electrode	
	F (DF)	p	F (DF)	p
Frequency	316.01 (2)	≤ 0.05	163.14 (2)	≤ 0.05
Displacement	9.83 (2)	≤ 0.05	4.09 (2)	≤ 0.05
pBWS	118.23 (2)	≤ 0.05	20.52 (2)	≤ 0.05
Muscle Group	57.47 (3)	≤ 0.05	17.33 (1)	≤ 0.05
Frequency \times pBWS	13.07 (4)	≤ 0.05	7.08 (4)	≤ 0.05
Frequency \times Muscle Group	8.89 (6)	≤ 0.05	1.38 (2)	$= 0.25$
Displacement \times Muscle Group	5.07 (6)	≤ 0.05	0.26 (2)	$= 0.75$
pBWS \times Muscle Group	15.73 (6)	≤ 0.05	29.57 (2)	≤ 0.05

Artefact Filter Validation

Randomly generated, band-limited (20 - 450hz), Gaussian white noise (Data) is shown in Figure 5.9(a). This signal is used in lieu of real EMG data in this example. A 27Hz sinusoidal signal with an amplitude equal to the average amplitude of the white-noise was generated and added to the artificial EMG data. Harmonics (2 - 18) of the 27Hz sinusoid with exponentially decreasing amplitude were then added to the artificial EMG also Figure 5.9(b). This new signal is representative of an EMG superimposed with noise analogous to a vibration artefact (Data + Artefact). This artificial data was used to investigate the efficacy of the stop-band filters used in this study to eliminate vibration artefact from real EMG data. Artificial data was used in this first instance because the true power of the artificial EMG and the artefact was known.

The effect of artefact on the original EMG signal was evident when it was viewed in the time domain, where a significant increase in the amplitude of the EMG signal was seen. Furthermore, the presence of the artefact was confirmed when viewing the data in the frequency domain. Sharp ‘spikes’ in signal power were observed at the harmonic frequencies. Filtering this data using the processes described in Sections 4.2.3 & 4.2.5, and specifically targeting the first seven harmonics only, resulted in a reduction in the amplitude of the signal to pre-artefact levels and a complete removal of the artefact spikes from the frequency plots.

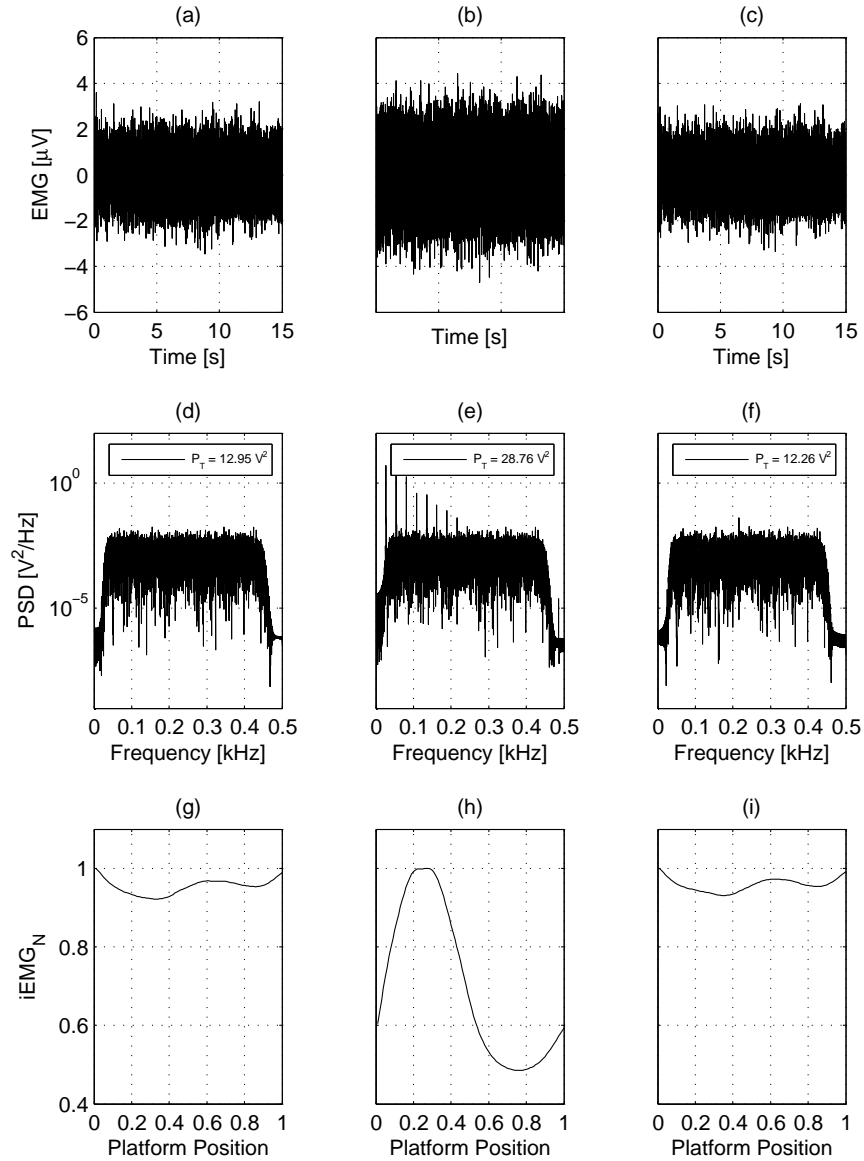


Figure 5.9: Artificially generated EMG data is shown in the left column in the time (a) and frequency domains (d). The same data with added noise is shown in the middle column (b) & (e). The noisy data was filtered and shown in the right column (c) & (f). The bottom row (g), (h) & (i) shows the results of a vibration-cycle analysis for each of these data sets (as described in Section 4.2.10). Data: Band-limited gaussian white noise; Data + Artefact: Data with added sinusoidal signal; Filtered Data: Filtered Data + Artefact, specifically removing sinusoidal signals; PSD: Power spectral density; P_T : Sum of PSD; iEMG: Interpolated EMG, that has been normalised to the maximum computed value.

This result was quantified by integrating the PSD data shown in Figure 5.9(c), (d) and (e). This provided an indication of the energy contained within the EMG. Integrating the PSD of the original data (Figure 5.9(a)) between 0 - 500Hz yielded a total signal energy of $12.95V^2$. The same procedure applied to the data with artefact (Figure 5.9(b)) yielded a total signal energy of $28.76V^2$. Therefore, the artefact increased the energy of the original signal by:

$$\frac{28.76 - 12.95}{12.95} \times \frac{100}{1} = 122.1\%$$

Specifically removing the first seven harmonics from this data using digital stop-band filters yielded the data shown in (Figure 5.9(c)). Integrating the power of this signal between 0 - 500Hz yielded a total signal energy of $12.26V^2$. The difference between this and the original signal was:

$$\frac{12.26 - 12.95}{12.95} \times \frac{100}{1} = -5.3\%$$

It is known that some of the harmonic signals (h_{8-18}) were still present in this data after filtering. Nonetheless, the energy of the filtered signal was 5.3% smaller than that of the original signal. This occurred because the filters almost completely removed all frequencies within their stop-band range. Some of the frequencies which were removed would have been genuine signal which coincidentally occurred within the same range as the artefact frequencies. Therefore eliminating these genuine signals has the effect of reducing the amplitude of the signal to a level smaller than the original.

The final observable effect of artefact on the EMG was seen when the EMG Vibration Cycle Analysis (Section 4.2.10) was applied to this artificial data. The data in this example was taken to be representative of EMG data acquired during the 27Hz vibration frequency condition. Purely Gaussian white noise with an amplitude of 1 is seen in Figure 5.9(g) as expected. The addition of a sinusoidal signal artefact to this signal is clearly seen to dominate (Figure 5.9(h)), therefore hiding the underlying trends in the data. Importantly, filtering the data returned the signal to its original shape (Figure 5.9(i)). This is analysed in further detail in Section 5.1.6 using real EMG data.

It was concluded from this analysis that a series of Butterworth stop-band filters were appropriate for the targeted and specific removal of artefact signals from EMG data acquired during WBV. Furthermore, this analysis highlighted that these stop-band filters will eliminate genuine EMG activity which occurs coincidentally at the same frequencies as the artefact signals.

5.1.3 EMG Classification

A total of 6480 unique EMG recordings were acquired using the active electrodes, filtered offline according to the standard ISEK guidelines, filtered again to remove vibration artefact and classified according to the methods described in Section 4.2.8. The variability of each EMG was then estimated by computing the Burst-to-Tonic Ratio (BTR). EMG data was classified as either an outlier or retained for further analysis using the criteria described in Table 4.5. The rejection of outlying data was required because in some instances it was identified that test subjects would lose balance during testing. This in turn induced additional

neuromuscular activity into the EMG that was unrelated to that caused by the vibration. The inclusion of this additional neuromuscular activity would result in an erroneous overestimation of muscle activity being attributed to WBV.

162 (2.5%) EMG were classified as outliers and rejected. The origins of the rejected EMG are summarised in Table 5.2. The majority of the rejected EMG were recorded from the TA with an increasing trend in the number of EMG rejected with increasing vibration frequency and vibration amplitude. This is reflected by a similar trend identified for increasing platform acceleration where a larger number of the rejected EMG were for higher levels of platform acceleration (data not shown in table). There was a reasonably even distribution in the number of EMG rejected across the different levels of pBWS.

Table 5.2: Breakdown of EMG outlier origins. TA = Tibialis Anterior; M-GA = Medial Gastrocnemius; VL = Vastus Lateralis; BF = Biceps Femoris.

Parameter	Composition of Rejected Values			
Muscle	TA	M-GA	VL	BF
	58.8%	15.2%	12.7%	13.3%
Frequency	12Hz	20Hz	27Hz	
	23.6%	33.9%	42.4%	
Displacement	1mm	2mm	3mm	
	26.7%	33.3%	40.0%	
pBWS	0%	50%	100%	
	47.9%	20.6%	31.5%	
Epoch	pre-Vib	Vib	post-Vib	
	23.6%	28.5%	47.9%	

All of these EMG were classified as outliers because the BTR exceeded 5. This was indicative of a highly variable signal which was not consistent with the neuromuscular responses normally found under the same experimental conditions. The average BTR of the included EMG was typically between 0.5 - 1, and a breakdown of these values found are shown in Figure 5.10 and 5.11. The BTR was larger during vibration than without as expected, and there were no differences between the results from the first and second tests of the same experimental conditions.

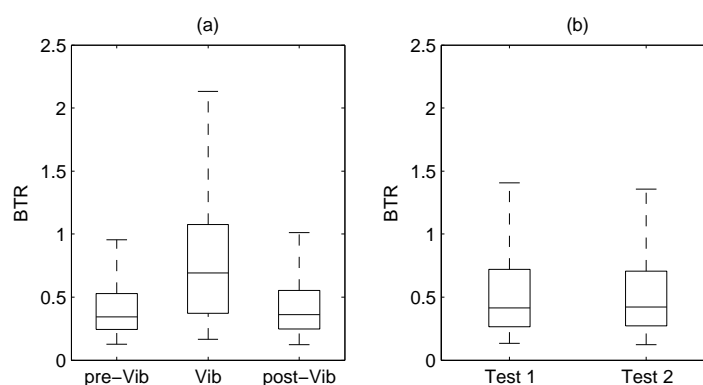


Figure 5.10: Summary of BTR results for the EMG included for further analysis. The data presented are for all subjects and test conditions and are grouped according to epoch (a) and test number (b).

The BTR increased with both vibration frequency and vibration amplitude. Furthermore, there was a decreasing trend in BTR values with increasing levels of pBWS. Typical values for the M-GA and VL were larger than those found for the TA and BF.

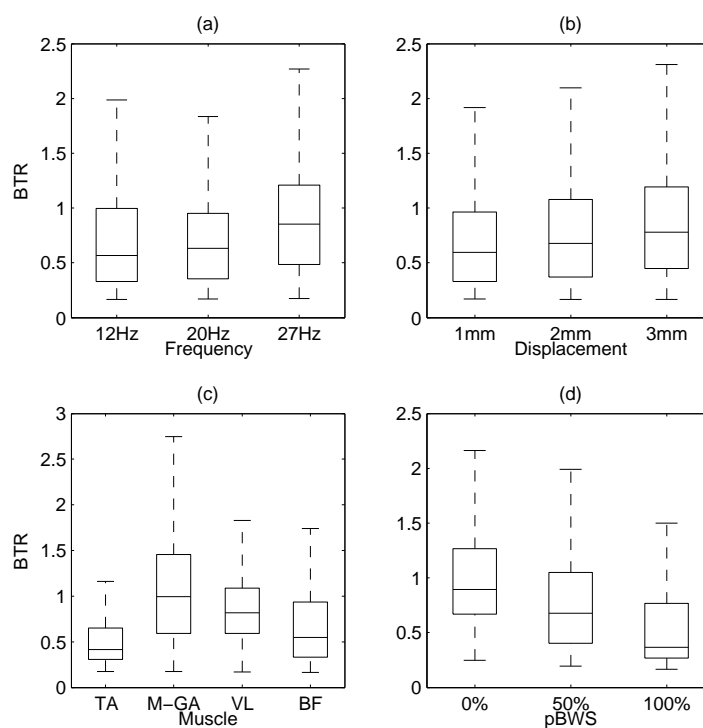


Figure 5.11: Overall summary of BTR results recorded during the vibration epoch only and included for further analysis. The data presented are for all subjects and testing sessions, and are grouped according to frequency (a), displacement (b), muscle group (c), and level of pBWS (d).

5.1.4 General EMG Amplitude Response

The intention of this initial amplitude analysis was to describe the general underlying trends of the neuromuscular response to WBV. A specific analysis is shown in the next section which investigates the neuromuscular response at specific combinations of vibration frequency, vibration amplitude and level of pBWS. To do this, it was first necessary to establish if WBV caused any increase in neuromuscular activity. The difference in response to different testing conditions (vibration frequency, vibration amplitude and level of pBWS) was investigated next. Finally, it was established if there was a difference in response of the different muscle groups to WBV.

General Results

Typical normalised EMG (EMG_N) (Equation (4.11)) values are shown in Figure 5.12 and the results of the statistical analysis of this grouped data is shown in Table 5.3. EMG_N was significantly different for all three epochs of the testing protocol: pre-Vibration, Vibration, and post-Vibration, but with the largest amount of activity occurring during vibration as expected. There was no statistically significant difference between the first and second testing sessions.

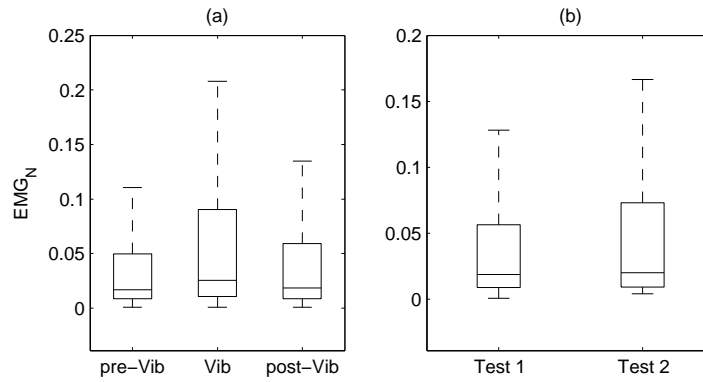


Figure 5.12: Overall summary of EMG results not rejected. Data for all subjects and test conditions are presented and grouped according to epoch and testing sessions.

Statistical results were based on a multi-way ANOVA for all EMG not rejected by the BTR threshold. The independent grouping factors were epoch and test condition. A post-hoc multiple comparisons procedure with Bonferroni correction was used to identify significantly different pairs when significance was identified by the ANOVA.

Table 5.3: General summary of statistical results. F: ANOVA F-statistic; DF: Degrees of Freedom; p: P-value; Gu_{Gl} : Groups identified to be significantly different in post-hoc analysis; Gu_{GL} : Paired data groups whose difference was statistically significant; Gu represents the grouped data with the larger value and Gl represents the grouped data with the smaller value; †: All grouped pairs found to be significantly different at $p \leq 0.05$.

Parameter	EMG_N		
	F (DF)	p	Gu_{Gl}
Epoch	187.2 (2)	≤ 0.01	†
Test	0.749 (1)	$= 0.387$	-

Vibration Results

It was concluded that vibration did cause an increase in neuromuscular activity and that this increase was statistically significant. This increase was therefore evaluated in relation to: (i) vibration frequency, (ii) vibration amplitude, (iii) level of pBWS, and (iv) the specific muscle group. The relative change in activity (ΔEMG) (Equation (4.12)) was calculated by comparing neuromuscular activity recorded during Vibration to baseline. Baseline was taken to be the pre-Vibration epoch in this instance. These data are shown with the corresponding values of EMG_N in Figure 5.13. The results of the supporting statistical analysis is shown in Table 5.4.

Statistical results were based on a multi-way ANOVA for all EMG which met the BTR inclusion criteria and were recorded during the Vibration epoch. The analysed data are for all subjects and test conditions, and the independent grouping factors were vibration frequency, vibration amplitude, muscle group, and level of pBWS. A post-hoc multiple comparisons procedure with a Bonferroni correction was used to identify significantly different pairs when overall statistical significance was indicated by the ANOVA.

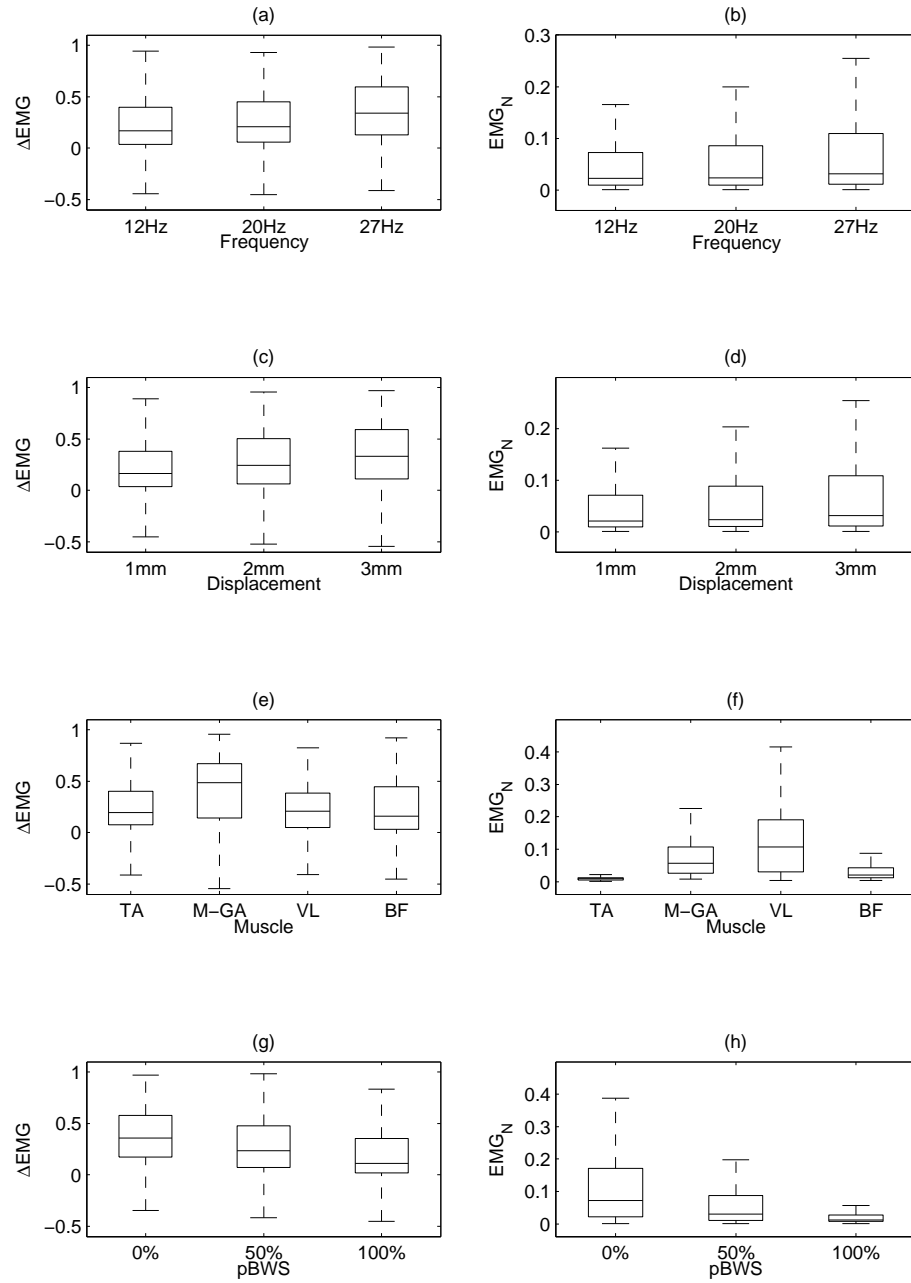


Figure 5.13: Summary of EMG amplitude response analysis of data acquired during the Vibration epoch only. The data presented in the left column ((a), (c), (e) & (g)) are ΔEMG and the data presented in the right column ((b), (d), (f) & (h)) are EMG_N . The data are for all subjects and tests, and are grouped according to: vibration frequency ((a) & (b)), vibration amplitude ((c) & (d)), muscle group ((e) & (f)), and level of pBWS ((g) & (h)).

Neuromuscular activity increased with both vibration frequency and vibration amplitude, and decreased with level of pBWS. Changes in ΔEMG between 15 and 30% were found for vibration frequency, with corresponding average EMG_N values between 2 and 3%. These changes were statistically significant and a post-hoc analysis showed that vibration at 27Hz resulted in a significantly larger increase in neuromuscular activity when compared to those changes that occurred at 12Hz. Similar values were found for vibration displacement and in each case the changes in EMG_N and ΔEMG were significant. Average values of EMG_N were between 1 and 7% depending on the level of pBWS and these changes were significant also. The corresponding values of ΔEMG decreased with increasing levels of pBWS and typical values of ΔEMG were between 10 and 35%. The post-hoc analysis showed that ΔEMG was significantly larger at 0% pBWS than at 50 or 100% pBWS.

It was observed that EMG_N was considerably different for each of the different muscle groups during vibration, and that these difference were statistically significant. The VL was most active during Vibration with typical values of approximately 10% MVC occurring. EMG_N of the M-GA was next with typical values of 5% MVC. The activity of the TA and BF were smallest and were activated to approximately 1 - 2% MVC. The trend in ΔEMG differed to this however. Comparatively, the largest relative changes to baseline were in the M-GA with average ΔEMG values of 50%. Changes compared to baseline for the TA, VL and BF were 20, 20, and 15% respectively. ΔEMG for M-GA was significantly larger than those change which occurred in the other muscle groups.

Table 5.4: General summary of WBV statistical results. ΔEMG : Change in EMG during Vibration with respect to pre-Vibration condition; EMG_N : EMG during vibration normalised to a MVC; F: ANOVA F-statistic; DF: Degrees of Freedom; p: P-value; Gu_{GL} : Groups identified to be significantly different in post-hoc analysis; Gu_{GL} : Paired data groups whose difference was statistically significant; Gu represents the grouped data with the larger value and Gl represents the grouped data with the smaller value; †: All pairs found to be significantly different at $p \leq 0.05$.

Parameter	ΔEMG			EMG_N		
	F (DF)	p	Gu_{GL}	F (DF)	p	Gu_{GL}
Frequency	64.66 (2)	≤ 0.01	†	30.94 (2)	≤ 0.01	$27_{12}; 27_{20}$
Displacement	64.06 (2)	≤ 0.01	†	30.90 (2)	≤ 0.01	†
Muscle	90.21 (3)	≤ 0.01	M-GA _{TA} ; M-GA _{VL} ; M-GA _{BF}	401.3 (4)	≤ 0.01	†
pBWS	76.96 (2)	≤ 0.01	†	350.1 (2)	≤ 0.01	†

5.1.5 Specific EMG Amplitude Response

Analysis of the acceleration data indicated that damping of the vibration was occurring as it was transmitted through the body, and that the transmission of vibration was degraded further as the level of pBWS was increased. The general EMG analysis confirmed that WBV elicited an increased neuromuscular response and that vibration frequency, vibration amplitude and level of pBWS each had a significant effect upon that response. The analysis also highlighted that the response across the different muscles was not uniform for a given set of experimental conditions. It was thus confirmed that the initial conditions for each

muscle group must be different. It was hypothesised that factors such as pBWS (which influences muscle tension) and the relative proximity of the muscle to the vibration platform (which influences vibration transmission) may each play a central role in the magnitude of the neuromuscular response during WBV.

It was confirmed that the proximity of the muscle to the vibration platform affected the magnitude of the response during WBV. The relationship between the existing contraction in the muscle and the magnitude of the neuromuscular response thereafter (during the application of WBV) was assessed by comparing EMG_N for the pre-Vibration and Vibration epochs. The scatter plots in Figure 5.14 show this relationship for each muscle respectively. A best fit trend line was overlaid the scatter plot to highlight the general trends and Pearson's correlation coefficient was used to quantify the relationship. The results of this statistical analysis are reported in Table 5.5.

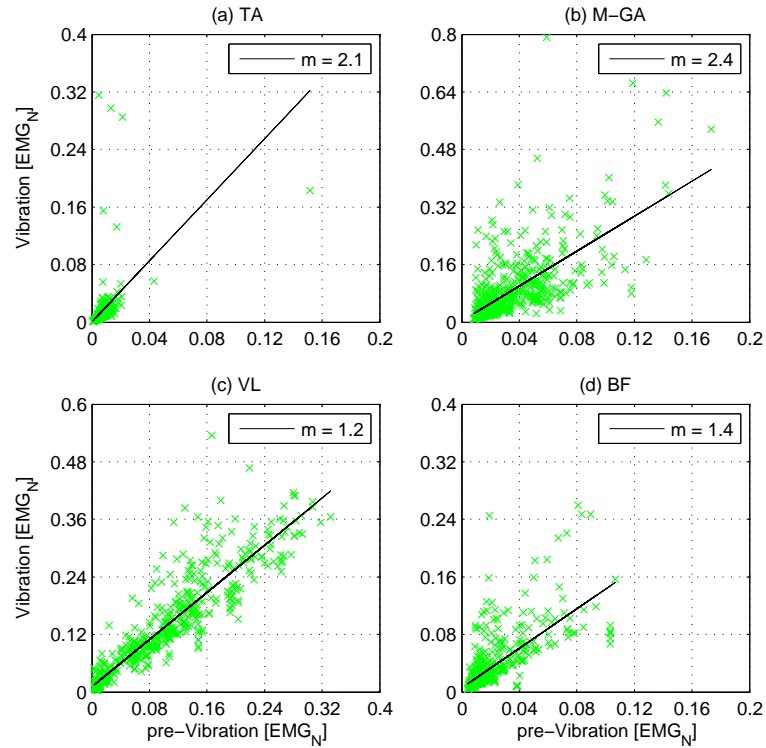


Figure 5.14: Scatter plot of pre-Vibration and Vibration muscle activity with best-fit trend line. The best-fit line assessed using Pearson's correlation coefficient and the slope of the line is shown on each subplot. Note that a slope (m) < 1 indicates an attenuation of neuromuscular activity during the application of WBV. TA: Tibialis Anterior, M-GA: Medial Gastrocnemius, VL: Vastus Lateralis, BF: Biceps Femoris.

The slope of the each trend line provides an general indication of the increase in muscle activity expected to occur during WBV. The slopes of the fitted lines were different in each case and the trends for the TA and M-GA were larger than those for the VL and BF. This evidence supports the hypothesis that magnitude of neuromuscular response was different for

each individual muscle group and that the proximity of the muscle to the source of vibration had a determinant role in the magnitude of the response. It was concluded that the amplitude response of each individual muscle group be assessed separately based on these two pieces of evidence.

Table 5.5: Trend-line statistics describing relationship between pre-Vibration and Vibration muscle activity. Best-fit line assessed using Pearson's correlation coefficient; R-value: Pearson's best fit correlation.

Muscle	Slope	Intercept	R-value	P-value
TA	2.1	0.0	0.2	<0.01
M-GA	2.4	0.0	0.4	<0.01
VL	1.2	0.0	0.8	<0.01
BF	1.4	0.0	0.5	<0.01

It was next determined that the initial muscle tension was different for each muscle group by comparing the magnitudes of neuromuscular activity (EMG_N) during the pre-Vibration condition. pre-Vibration was considered analogous to a vibration frequency of 0Hz and vibration amplitude of 0mm and is described otherwise as the 0.0g condition. The magnitude of neuromuscular activity during the 0.0g condition is plotted for each muscle in Figures 5.15 - 5.18 below. It can clearly be seen that the magnitude of EMG_N decreases with each increasing increment in the level of pBWS at the 0.0g condition. Furthermore, the baseline underlying level of neuromuscular activity in each muscle group was different.

The amplitude response of each individual muscle to WBV is presented in Figures 5.15-5.18 where the data is presented in three ways: (i) by describing the absolute measured values (this data is not used for further discussion in this chapter but is provided for subsequent comparison with SCI data); (ii) by describing the magnitude of neuromuscular activity relative to MVC; and (iii) by describing the relative change in neuromuscular activity when compared to a baseline condition. The presented data are for all EMG that were not rejected by the criterion set out in Section 4.2.8. The data were grouped according to level of pBWS and platform acceleration and the 0.0g condition represents baseline data. The specific vibration frequency and vibration amplitude conditions associated with each data group may be determined by referencing the acceleration values described on the x-axis to those outlined in Table 4.6. A multi-way ANOVA was carried out on all EMG_N and ΔEMG data at each level of pBWS. The independent grouping factors were vibration frequency and vibration amplitude and the results of the statistical analysis are presented in Tables 5.6-5.9.

Tibialis Anterior

WBV caused an increase in TA neuromuscular activity. ΔEMG increased with platform acceleration with the largest change occurring at 0% pBWS and smallest at 100% pBWS. ΔEMG was typically between 20 - 50% at 0% pBWS, which equated to comparatively small EMG_N of 1 - 2%. ΔEMG at 50 and 100% pBWS were broadly similar, where increases between 10 - 30% were found. This equated to an EMG_N of approximately 1%. The results of the statistical of these findings are outlined in Table 5.6.

Table 5.6: Summary of the statistical analysis of TA amplitude response results. A multi-way ANOVA was used to identify significant changes in EMG_N and ΔEMG . The independent grouping factors were vibration frequency and vibration amplitude. The interaction between these terms was also investigated. EMG_N : EMG normalised to a MVC (Equation (4.12)); ΔEMG : Change in activity relative to baseline (Equation (4.12)); TA: Tibialis anterior; pBWS: Partial body weight support; F: ANOVA F-statistic; ANOVA: Analysis of Variance; DF: Degrees of freedom; p: P-value; Gu_{GL} : Paired data groups whose difference was statistically significant; Gu represents the grouped data with the larger value and Gl represents the grouped data with the smaller value.

pBWS	Parameter	ΔEMG			EMG_N		
		F (DF)	p	Gu_{Gl}	F (DF)	p	Gu_{Gl}
0%	Frequency	14.60 (2)	≤ 0.01	$27_{12}; 27_{20}$	0.98 (2)	$=0.35$	-
	Displacement	12.01 (2)	≤ 0.01	$3_1; 3_2$	3.97 (2)	$=0.08$	-
	Interaction	0.32 (4)	$=0.84$	-	1.13 (4)	$=0.34$	-
50%	Frequency	6.47 (2)	≤ 0.05	$27_{12}; 27_{20}$	1.25 (2)	$=0.29$	-
	Displacement	13.18 (2)	≤ 0.01	$3_1; 3_2$	0.52 (2)	$=0.49$	-
	Interaction	0.64 (4)	$=0.63$	-	0.94 (4)	$=0.36$	-
100%	Frequency	3.75 (2)	$=0.05$	27_{12}	0.08 (2)	$=0.78$	-
	Displacement	2.34 (2)	$=0.14$	-	0.70 (2)	$=0.43$	-
	Interaction	0.79 (4)	$=0.52$	-	0.96 (4)	$=0.35$	-

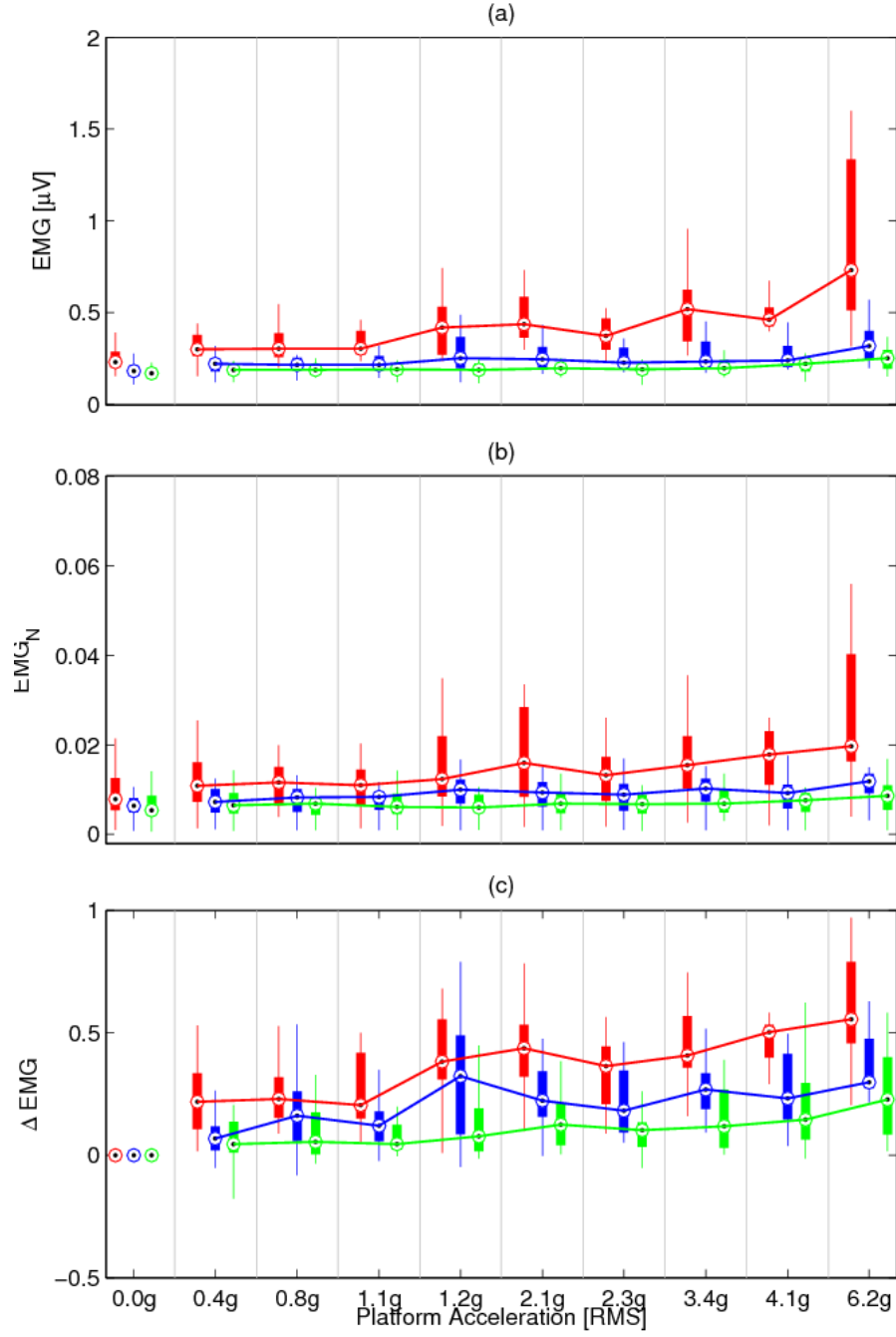


Figure 5.15: Amplitude response of TA to WBV. The neuromuscular response is presented as EMG (a), EMG_N (b) and Δ EMG (c). The presented data are for all subjects and test conditions. The data were grouped according the platform acceleration (Equation (4.2)) and level of pBWS. Pre-Vibration data is presented for reference purposes and can be identified as the 0.0g condition. The specific vibration and amplitude conditions used are identifiable by cross-referencing the acceleration values presented to those described in Table 4.6.

Medial Gastrocnemius

Larger increases in ΔEMG were found for the M-GA. These increases were largest at 0% pBWS and smallest at 100% pBWS. This trend is consistent with that found for the TA. ΔEMG was broadly similar at the 0 and 50% pBWS conditions, where typical values were between 25 - 70%. This equated to a typical EMG_N of 6 - 20% MVC at 0% pBWS, and 3 - 10% MVC at 50% pBWS. ΔEMG was between 5 and 60% which equated to an EMG_N of 1 - 7% MVC at 100% pBWS. These increases were statistically significant in many cases and the results of the statistical analysis are outlined in Table 5.7.

Table 5.7: Summary of the statistical analysis of M-GA amplitude response results. A multi-way ANOVA was used to identify significant changes in EMG_N and ΔEMG . The independent factors used are vibration frequency and vibration amplitude. The interaction between these terms was also investigated. EMG_N : EMG normalised to a MVC (Equation (4.12)); ΔEMG : Change in activity relative to baseline (Equation (4.12)); M-GA: Medial gastrocnemius; pBWS: Partial body weight support; F: ANOVA F-statistic; ANOVA: Analysis of Variance; DF: Degrees of freedom; p: P-value; Gu_{GL} : Paired data groups whose difference was statistically significant; Gu represents the grouped data with the larger value and Gl represents the grouped data with the smaller value; †: All grouped data were significantly different from one another.

pBWS	Parameter	ΔEMG			EMG_N		
		F (DF)	p	Gu_{GL}	F (DF)	p	Gu_{GL}
0%	Frequency	21.33 (2)	≤ 0.01	†	16.69 (2)	≤ 0.01	$27_{12}; 27_{20}$
	Displacement	5.80 (2)	≤ 0.05	3_1	14.16 (2)	≤ 0.01	$3_2; 3_1$
	Interaction	0.21 (4)	$= 0.90$	-	1.17 (4)	$= 0.34$	-
50%	Frequency	6.39 (2)	≤ 0.01	$27_{12}; 27_{20}$	7.65 (2)	≤ 0.05	$27_{12}; 27_{20}$
	Displacement	12.48 (2)	≤ 0.01	$3_1; 2_1$	13.97 (2)	≤ 0.01	$3_2; 3_1$
	Interaction	0.31 (4)	$= 0.87$	-	1.03 (4)	$= 0.39$	-
100%	Frequency	10.08 (2)	≤ 0.01	$27_{12}; 27_{20}$	2.86 (2)	$= 0.10$	-
	Displacement	6.00 (2)	≤ 0.05	3_1	6.99 (2)	≤ 0.05	$3_2; 3_1$
	Interaction	0.41 (4)	$= 0.80$	-	1.24 (4)	$= 0.31$	-

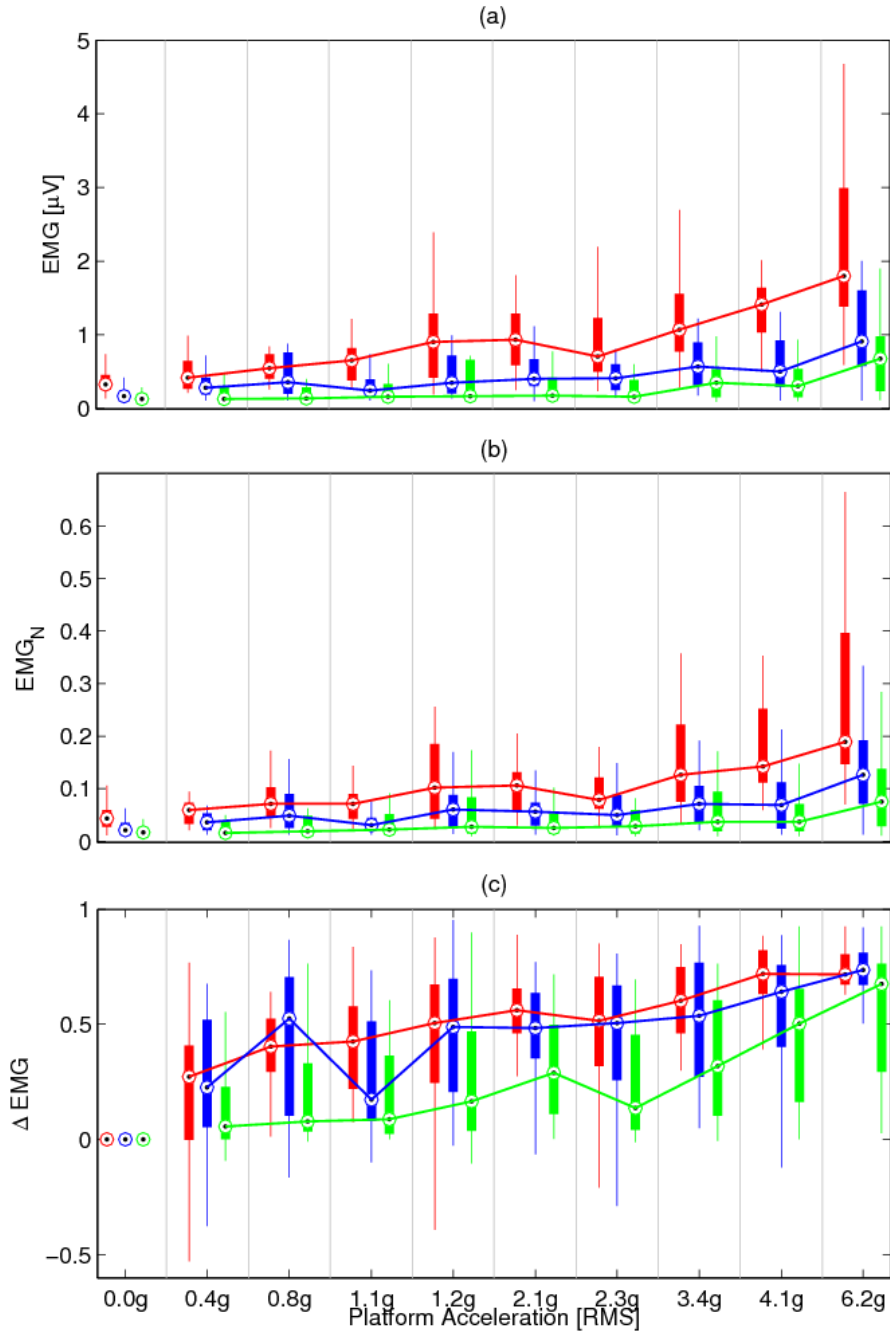


Figure 5.16: Amplitude response of M-GA to WBV. The neuromuscular response is presented as EMG (a), EMG_N (b) and Δ EMG (c). The presented data are for all subjects and test conditions. The data were grouped according the platform acceleration (Equation (4.2)) and level of pBWS. Pre-Vibration data is presented for reference purposes and can be identified as the 0.0g condition. The specific vibration and amplitude conditions used are identifiable by cross-referencing the acceleration values presented to those described in Table 4.6.

Vastus Lateralis

EMG_N of the VL was considerably different at each level of pBWS where typical values at 0% pBWS were between 18 - 28% MVC. Typical values at 50% pBWS were approximately

10% MVC and 1% MVC at 100% pBWS. ΔEMG was broadly similar across all levels of pBWS where typical values were between 5 and 30%. Results of the statistical analysis are presented in Table 5.8.

Table 5.8: Summary of the statistical analysis of VL amplitude response results. A multi-way ANOVA was used to identify significant changes in EMG_N and ΔEMG . The independent grouping factors were vibration frequency and vibration amplitude. The interaction between these terms was also investigated. EMG_N : EMG normalised to a MVC (Equation (4.12)); ΔEMG : Change in activity relative to baseline (Equation (4.12)); VL: Vastus lateralis; pBWS: Partial body weight support; F: ANOVA F-statistic; ANOVA: Analysis of Variance; DF: Degrees of freedom; p: P-value; Gu_{GL} : Paired data groups whose difference was statistically significant; Gu represents the grouped data with the larger value and Gl represents the grouped data with the smaller value.

pBWS	Parameter	ΔEMG			EMG_N		
		F (DF)	p	Gu_{GL}	F (DF)	p	Gu_{GL}
0%	Frequency	8.82 (2)	≤ 0.01	$27_{12}; 27_{20}$	2.75 (2)	$= 0.11$	-
	Displacement	2.74 (2)	$= 0.09$	-	1.18 (2)	$= 0.33$	-
	Interaction	0.03 (4)	$= 1.00$	-	0.09 (4)	$= 0.97$	-
50%	Frequency	0.23 (2)	$= 0.79$	-	0.55 (2)	$= 0.56$	-
	Displacement	2.64 (2)	$= 0.11$	-	1.05 (2)	$= 0.36$	-
	Interaction	0.46 (4)	$= 0.76$	-	0.09 (4)	$= 0.96$	-
100%	Frequency	0.45 (2)	$= 0.64$	-	0.08 (2)	$= 0.86$	-
	Displacement	0.18 (2)	$= 0.84$	-	0.46 (2)	$= 0.63$	-
	Interaction	0.29 (4)	$= 0.88$	-	0.08 (4)	$= 0.98$	-

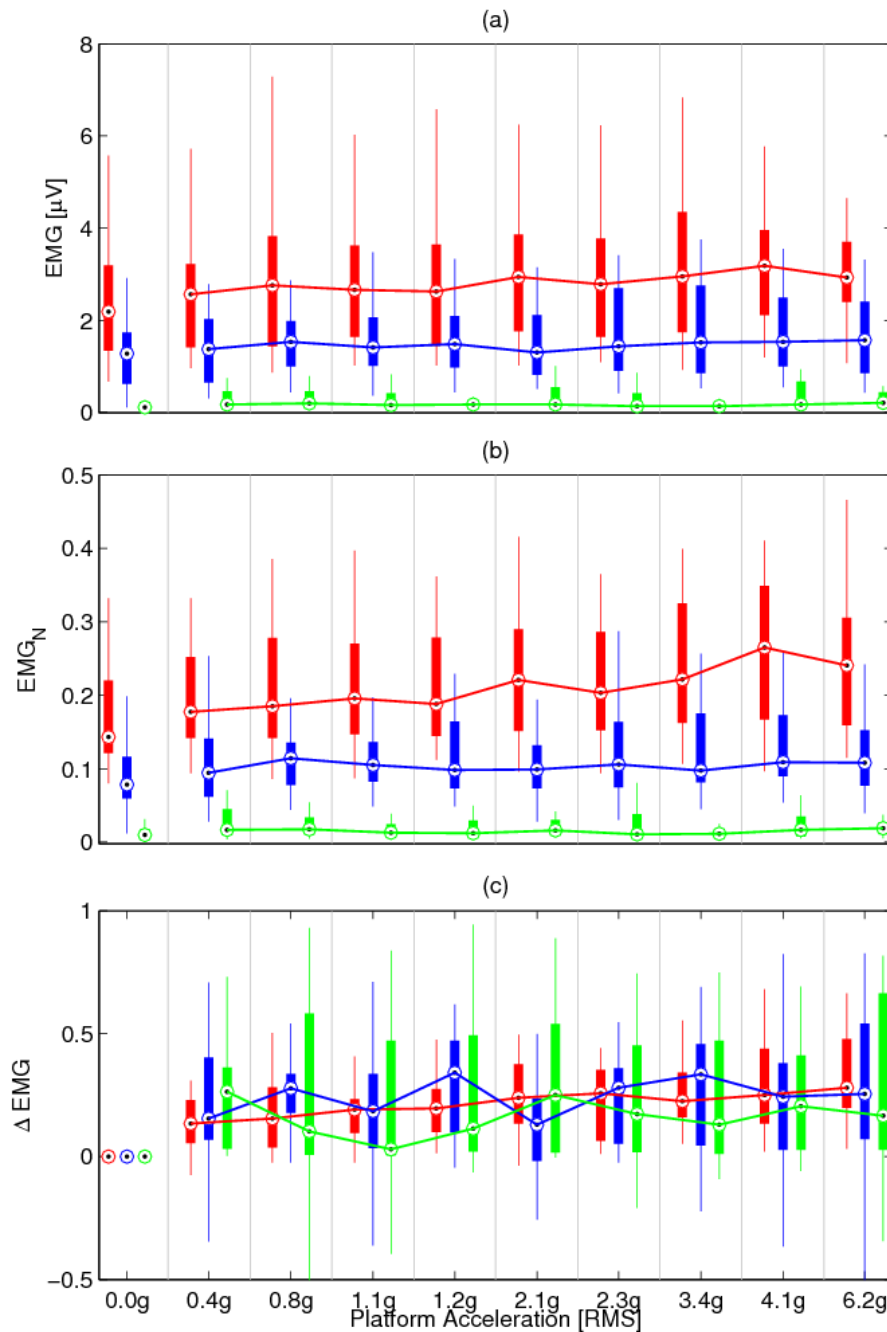


Figure 5.17: Amplitude response of VL to WBV. The neuromuscular response is presented as EMG (a), EMG_N (b) and ΔEMG (c). The presented data are for all subjects and test conditions. The data were grouped according the platform acceleration (Equation (4.2)) and level of pBWS. Pre-Vibration data is presented for reference purposes and can be identified as the 0.0g condition. The specific vibration and amplitude conditions used are identifiable by cross-referencing the acceleration values presented to those described in Table 4.6.

Biceps Femoris

The trends in ΔEMG for the BF were broadly similar to those found for the TA and M-GA where the levels of activity were largest at 0% pBWS and smallest at 100% pBWS. ΔEMG

was typically between 5 and 70% at 0% pBWS which equated to an EMG_N of 3 - 8% MVC. ΔEMG was between 3 - 35% at 50% pBWS and 3 - 8% at 100% pBWS. These increases equated to an absolute EMG_N of 1 - 2% MVC. The results of the statistical analysis are presented in Table 5.9.

Table 5.9: Summary of the statistical analysis of BF amplitude response results. A multi-way ANOVA was used to identify significant changes in EMG_N and ΔEMG . The independent grouping factors were vibration frequency and vibration amplitude. The interaction between these terms was also investigated. EMG_N : EMG normalised to a MVC (Equation (4.12)); ΔEMG : Change in activity relative to baseline (Equation (4.12)); BF: Biceps femoris; pBWS: Partial body weight support; F: ANOVA F-statistic; ANOVA: Analysis of Variance; DF: Degrees of freedom; p: P-value; Gu_{GL} : Paired data groups whose difference was statistically significant; Gu represents the grouped data with the larger value and Gl represents the grouped data with the smaller value.

pBWS	Parameter	ΔEMG			EMG_N		
		F (DF)	p	Gu_{Gl}	F (DF)	p	Gu_{Gl}
0%	Frequency	19.14 (2)	≤ 0.01	$27_{12}; 27_{20}$	10.51 (2)	≤ 0.01	$27_{12}; 27_{20}$
	Displacement	14.20 (2)	≤ 0.01	$3_1; 2_1$	4.88 (2)	≤ 0.05	3_1
	Interaction	0.13 (4)	$= 0.97$	-	0.40 (4)	$= 0.75$	-
50%	Frequency	3.06 (2)	$= 0.11$	-	2.28 (2)	$= 0.15$	-
	Displacement	2.08 (2)	$= 0.17$	-	0.35 (2)	$= 0.61$	-
	Interaction	0.39 (4)	$= 0.67$	-	0.34 (4)	$= 0.75$	-
100%	Frequency	0.22 (2)	$= 0.77$	-	0.36 (2)	$= 0.65$	-
	Displacement	2.41 (2)	$= 0.12$	-	0.28 (2)	$= 0.70$	-
	Interaction	0.54 (4)	$= 0.66$	-	0.09 (4)	$= 0.91$	-

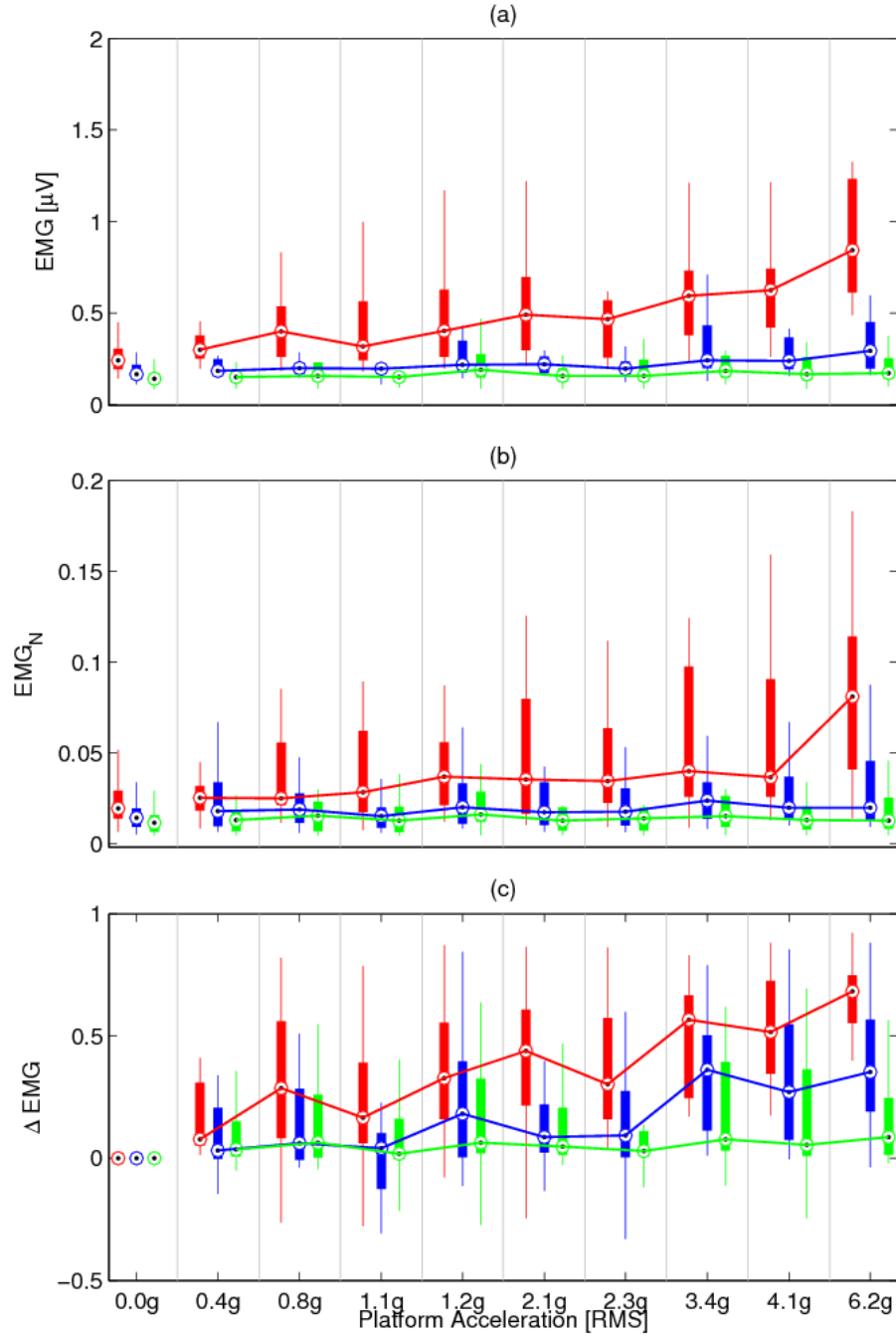


Figure 5.18: Amplitude response of BF to WBV. The neuromuscular response is presented as EMG (a), EMG_N (b) and Δ EMG (c). The presented data are for all subjects and test conditions. The data were grouped according the platform acceleration (Equation (4.2)) and level of pBWS (bottom panel). Pre-Vibration data is presented for reference purposes and can be identified as the 0.0g condition. The specific vibration and amplitude conditions used are identifiable by cross-referencing the acceleration values presented to those described in Table 4.6.

In summary this specific analysis supports the findings of the general analysis described in Section 5.1.4. Vibration frequency, vibration amplitude and pBWS did significantly affect the

magnitude of the neuromuscular response to WBV. The trends of increase in neuromuscular response were specific to each muscle group as expected. Both ΔEMG and EMG_N were generally largest at 0% pBWS and smallest at 100% pBWS. Furthermore, each of these parameters were typically found to increase with platform acceleration.

The TA and BF were the least active with similar values of ΔEMG and correspondingly showed the smallest EMG_N values. Conversely the M-GA and VL were the most active. EMG_N values in excess of 20% MVC were found for each muscle, however the corresponding ΔEMG values were considerably higher in the M-GA than the VL. These results further support the hypothesis that proximity (of the muscle to the source of vibration) and tension (the initial magnitude of voluntary muscle activity) each play a central role in determining the amplitude of the overall neuromuscular response to WBV.

5.1.6 Vibration Cycle Analysis

The Vibration Cycle Analysis methodology was described in detail in Section 4.2.10 and was qualified using an example which used artificially generated data that resembled a real EMG. The same qualification process was repeated in the case study below. Increases in the amplitude of the EMG which occurred at regular intervals during each vibration cycle were expected to be visible in the normalised EMG output from the analysis. It was established previously that vibration artefact also affected the shape of an EMG when viewed in this way. This effect was minimised with appropriate off-line filtering.

A regular increase in the magnitude of the EMG was found to occur over the course of a single vibration cycle in the case study described below and it was concluded that this increase was not related to vibration artefact. This finding is fundamentally different to that found for the artificial data (Section 5.1.2) for which all periodic changes in the EMG amplitude were eliminated by off-line filtering.

Case Study

The previous worked example (Section 4.2.10) anticipated the effect of vibration artefact on the shape of an EMG. The same analysis methodology was carried in this instance with real EMG data that was recorded during WBV. The results were plotted and are shown in Figure 5.19. The first plot is of an EMG which was recorded during the pre-Vibration epoch, the second EMG during WBV but with only basic filtering (Section 4.2.3) applied, and the third EMG is the same as the second but with the harmonic frequencies specifically removed also (Section 4.2.5).

Peaks at the vibration frequency and harmonics were found when the EMG was viewed in the frequency domain as expected. This was confirmed by viewing EMG recorded during WBV with only basic filtering applied. No spikes were present in the pre-Vibration EMG because vibration artefact can not occur in the absence of vibration, nor were any identified in the fully filtered Vibration EMG either. This finding eliminates any potential electrical source for the artefact and corroborates the hypothesis that the artefact was caused by the WBV. It was also finally found that the amplitude of the EMG recorded during WBV was reduced when the vibration artefact was removed.

A Vibration Cycle Analysis was carried out next and the results are plotted in Figure 5.19 also. No significant temporal changes were found for the first EMG. A temporal relationship was indicated in the second EMG, where a distinct shape and discernible peak in the EMG was identifiable. However, it was not clear if this observed change was the consequence of vibration artefact or regular and repeated increases in neuromuscular activity occurring at a particular point of the vibration cycle. A well defined peak in the EMG was present in the third fully filtered EMG though it was reduced in magnitude when compared to that for the second. This result indicates that a temporal relationship did indeed exist between the phase of vibration and the neuromuscular response, whereby a well defined and distinct increases in neuromuscular activity occurred at a specific time point during each single cycle of vibration.

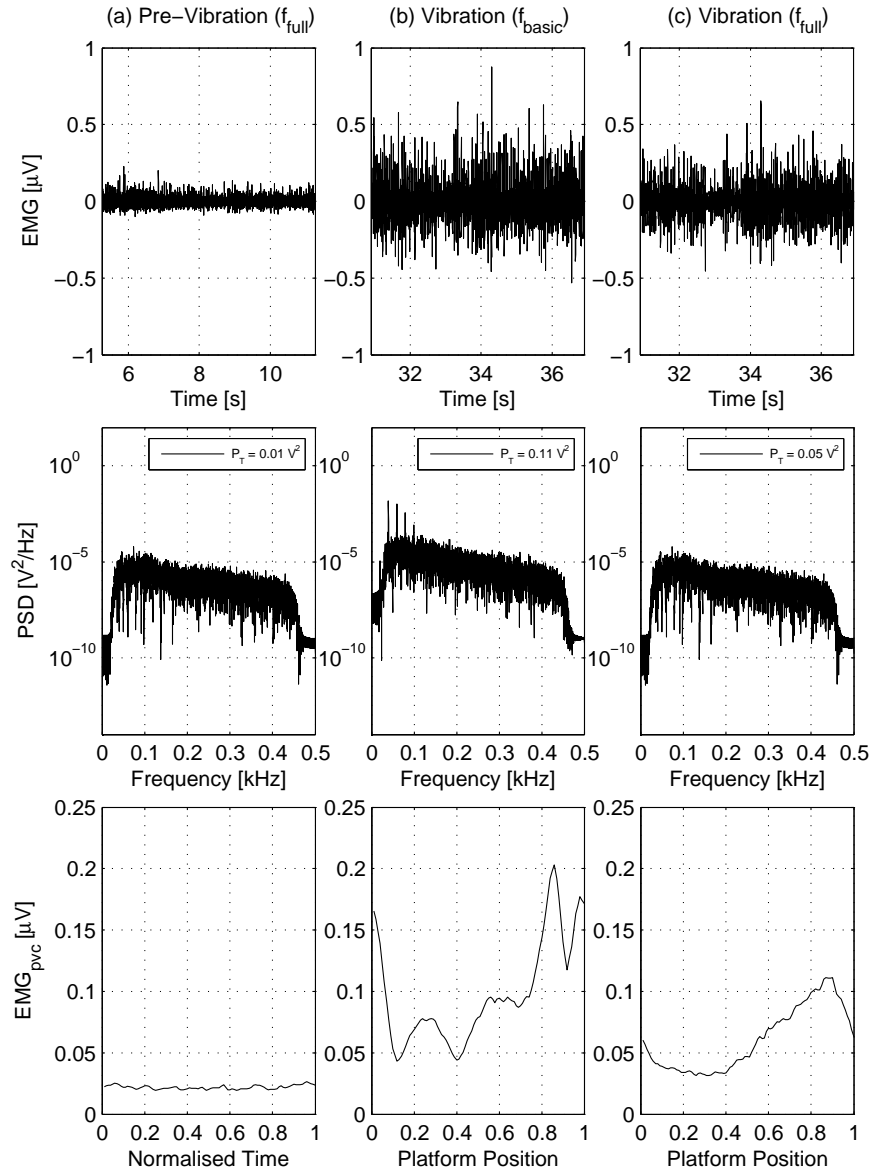


Figure 5.19: Results from a Vibration Cycle Analysis carried out using real EMG data recorded from Subject 4 during Test 1. Vibration was applied at a vibration frequency of 20Hz and a vibration amplitude of 2mm. No pBWS was provided. The data presented in column (a) are from the EMG recorded during the pre-Vibration epoch. The EMG was filtered according to the standards outlined in Sections 4.2.3 & 4.2.5. The data presented in column (b) are from the EMG recorded during the Vibration epoch. The EMG was filtered according to the standards outlined in Sections 4.2.3 only. The data presented in column (c) are from the EMG presented in column (b) but with additional filtering applied as described in Section 4.2.5. The data presented in the first row are the EMG presented in the time domain. The data presented in the second row are the same EMG presented in the frequency domain. The data presented in the third row are from the same EMG analysed using the vibration cycle analysis. f_{full} : Standard filtering with additional artefact removal according to the methods described in Sections 4.2.3 & 4.2.5; f_{basic} : Filtering according to the standards described in Section 4.2.3; P_T : Energy of the EMG when viewed in the frequency domain as calculated by integrating the PSD; PSD: Power Spectral Density; pvc: Per Vibration Cycle.

A Vibration Cycle Analysis was therefore carried out for every participant in this study to determine which parameter specifically elicited such a response. The results are outlined in the General Analysis below.

General Analysis

A Vibration Cycle Analysis was carried out on every EMG that met the inclusion criteria described in Section 4.2.8 and the results are summarised in Figure 5.20. The vibration cycle data from each test were averaged for all subjects, tests, and test epoch: pre-Vibration, Vibration, post-Vibration. The averaged data were then grouped according to: vibration frequency, vibration amplitude, muscle, and level of pBWS, and normalised to maximum. This was done to determine qualitatively which of these factors was most likely to influence a temporal relationship between neuromuscular activity and a single cycle of vibration.

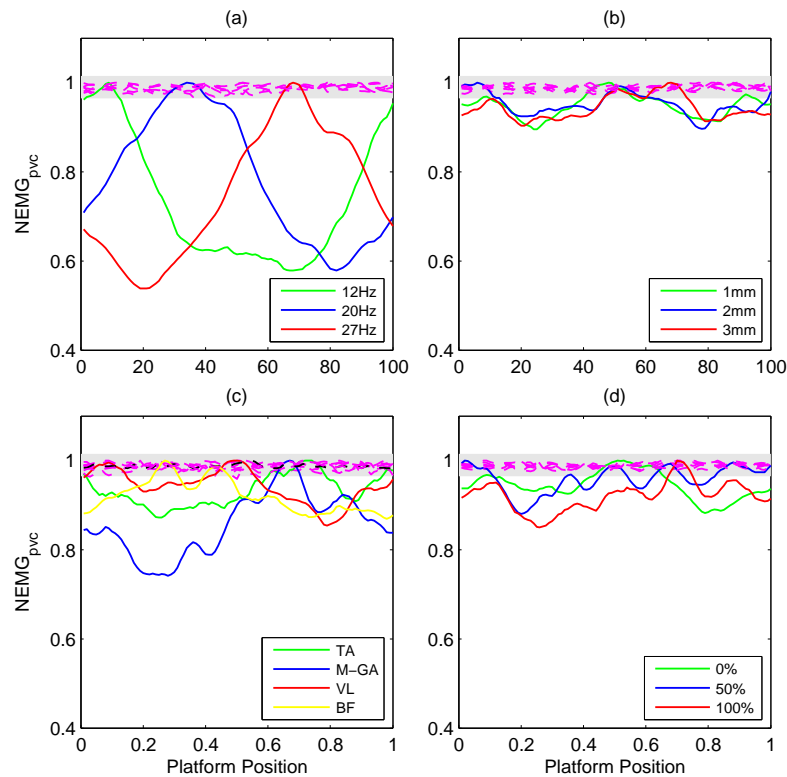


Figure 5.20: Summary of Vibration Cycle Analysis results. The presented data were derived from all of the recorded EMG that met the inclusion criteria outlined in Section 4.2.8. The results were grouped for all subjects, tests, and test epoch. Pre-, and post-Vibration data are indicated by dashed lines and highlighted by the grey band, and vibration data are indicated by solid lines. Depending on the factor being plotted, additional grouping factors included: vibration frequency (a), vibration amplitude (b), muscle (c), and level of pBWS (d). Grouped and averaged data were normalised to maximum. The sub-categories of each grouping factor are defined in the corresponding legends. NEMG_{pvc}: Per vibration cycle of the EMG normalised to its maximum.

No significant fluctuation or change in neuromuscular activity was observed for those EMG

recorded during the pre-, and post-Vibration epoch as expected and were therefore excluded from further analysis. Some fluctuation was found for the EMG grouped according to vibration amplitude and level of pBWS. However, these changes were relatively small when compared to those EMG which were grouped and averaged according to the muscle group and vibration frequency. It was thus hypothesised that: (i) the neuromuscular response was related to vibration frequency, and (ii) the response was specific to each muscle. These two grouping factors were therefore analysed in greater detail.

Specific Analysis

The Vibration Cycle Analysis was repeated and restricted to include those EMG which were recorded during the Vibration epoch only. Furthermore, the analysis included data only from those subjects whose output EMG from the first and second testing sessions were similar in shape as determined using Pearson's correlation coefficient. For example, the shape of the output EMG recorded from Subject 1 during Test 1 at the 20Hz, 1mm, 0% pBWS test condition needed to be similar to that recorded from the same subject and conditions during Test 2.

Pearson's correlation coefficient (r) was used to determine if the output EMG from each of the testing sessions were sufficiently 'similar'. Both output EMG were rejected if: (i) the correlation between the two EMG was non-significant ($p \geq 0.05$), or (ii) very poor or negative ($r \leq 0.1$). In the first version of this analysis an accepted correlation (r) of 0.7 or greater was set, which would indicate a high between test correlation. However, this limitation was found to be excessively restrictive with a small quantity of TA and M-GA data retained, and no VL or BF data retained. A lower accepted r -value of 0.1 was used instead and the analysis was re-run. The retained data were averaged for all subjects and grouped according to muscle group and vibration frequency and the results are shown in Figure 5.21.

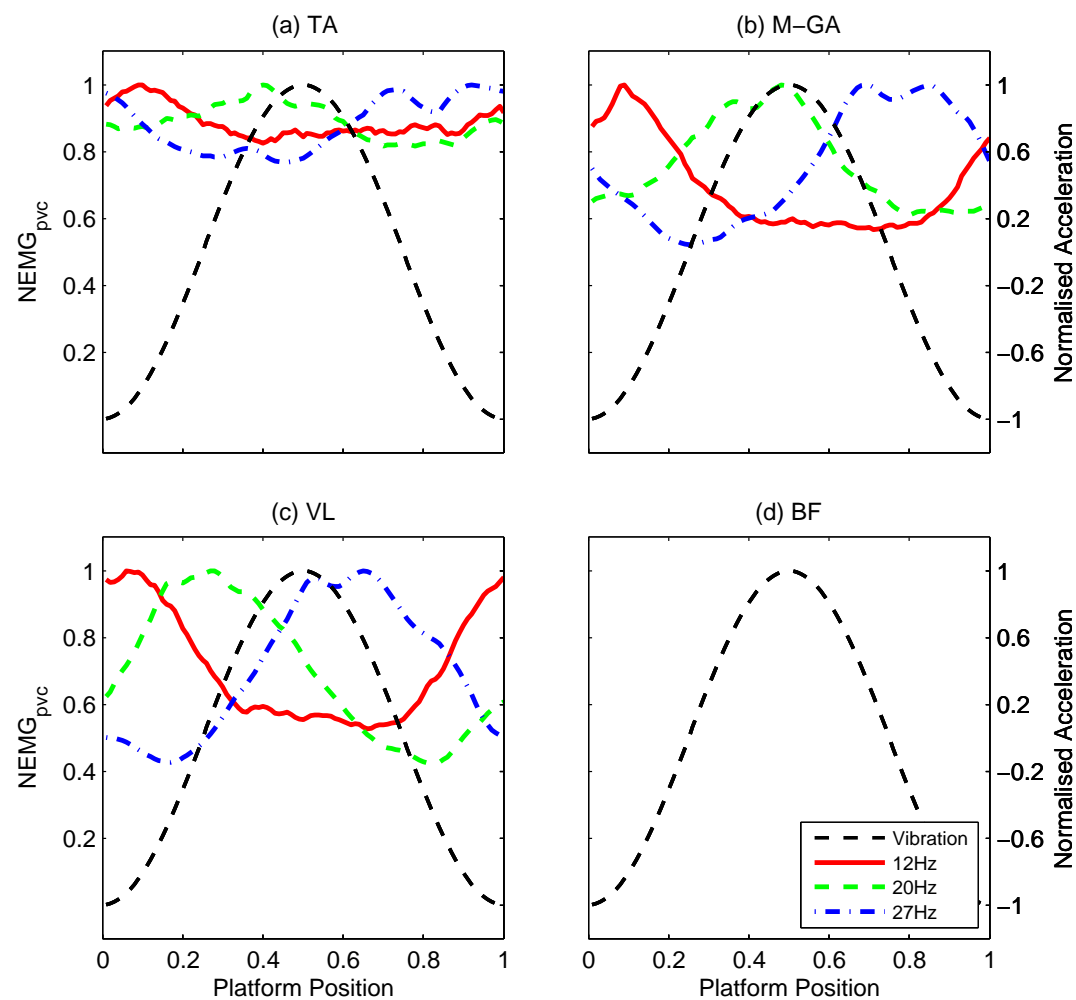


Figure 5.21: Results of vibration cycle analysis for EMG recorded during WBV only. The presented data were derived from the recorded EMG that met the inclusion criteria described in Section 4.2.8. The results were averaged for all subjects and tests, and grouped according to vibration frequency and muscle group. Grouped and averaged data were normalised to the respective maximum values. $NEMG_{pvc}$: Per vibration cycle of the EMG normalised to its maximum.

A distinct but broadband increase in the amplitude of the EMG was found. The point at which this distinct peak occurred was different for each muscle and vibration frequency respectively and the latencies of the occurrence of the peaks relative to the start of the vibration cycle are summarised in the table below.

Table 5.10: Summary of vibration cycle results. Data grouped and averaged for the Vibration epoch, muscle group, and vibration frequency. Peak values were identified using Figure 5.21 and the results are presented as the point during the vibration cycle at which the peak occurred. This point is presented as a percentage of completion of the vibration cycle where 0% is the beginning of the vibration cycle, and 100% is the end of the vibration cycle. No data is presented for the BF because no relationship was identified. F_{Vib} = Vibration frequency; TA = Tibialis Anterior; M-GA = Medial Gastrocnemius; VL = Vastus Lateralis; BF = Biceps Femoris.

Muscle	$F_{Vib} = 12\text{Hz}$	$F_{Vib} = 20\text{Hz}$	$F_{Vib} = 27\text{Hz}$
TA	9%	40%	82%
M-GA	9%	48%	77%
VL	7%	28%	60%

The occurrence of each of the peaks were broadly similar for the different participants at a given vibration frequency, but at a different latency from the beginning of the vibration cycle. It was hypothesised that a common stimulus may be eliciting this characteristic response and that the stimulus may be common for the different vibration periods. The latency between each peak and the preceding platform positions were therefore determined for each muscle group and vibration period. An example from the M-GA is plotted in Figure 5.22.

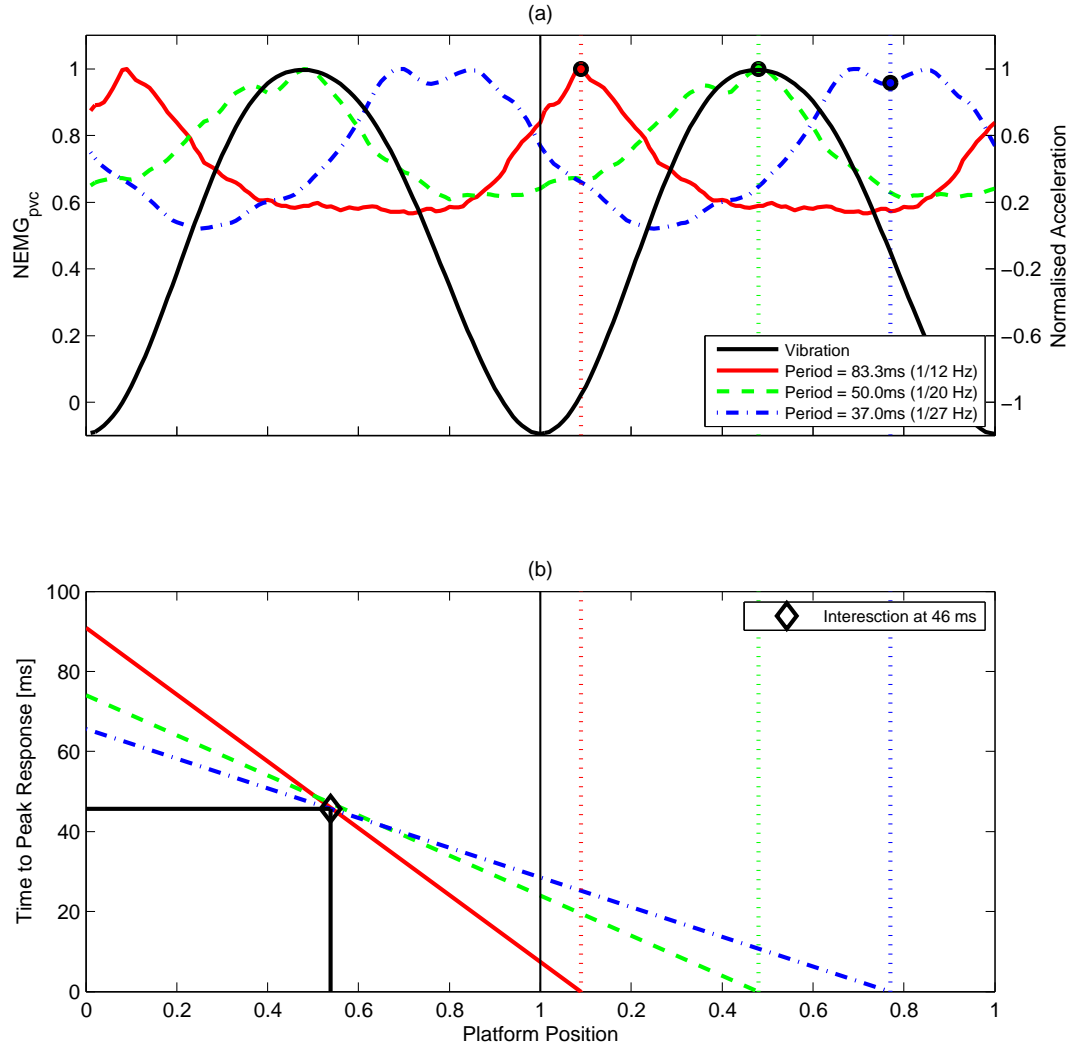


Figure 5.22: Outline of two consecutive vibration cycle EMG responses recorded from the M-GA. The vibration cycle EMG were analysed for each vibration period, normalised to maximum ($NEMG_{pvc}$) and plotted against platform position, where 0 indicates the beginning of a vibration cycle and 1 indicates the end of the same vibration cycle. The $NEMG_{pvc}$ recorded at a vibration frequency of 12Hz (83ms) is indicated by a solid line (-), the $NEMG_{pvc}$ recorded at a vibration frequency of 20Hz (50ms) is indicated by a dashed line (- -), and the $NEMG_{pvc}$ recorded at a vibration frequency of 27 Hz (37ms) is indicated by a dash-dot (- .). The relative amplitudes of each $NEMG_{pvc}$ are plotted against the primary y-axis (a). Vertical platform acceleration was also normalised to maximum and plotted against platform position. The amplitude of normalised acceleration is plotted against the secondary y-axis (a). The solid vertical line indicates the transition from one vibration cycle to the next. The dashed vertical lines indicate the peaks in each $NEMG_{pvc}$ and highlight the relative difference in their occurrence relative to each other. The latency of each of peak response relative to the preceding platform positions were determined and are indicated by the straight lines plotted in (b), where the slope of each line is equivalent to the corresponding vibration period (i.e. 83, 50, & 37ms). Convergence of the lines occurred at approximately 0.54 completion of the previous vibration cycle, and is equivalent to a latency of 46ms for all vibration frequencies. M-GA: Medial-Gastrocnemius, $NEMG_{pvc}$: Normalised EMG Per Vibration Cycle.

The results from Figure 5.22 indicated that a common stimulus did indeed exist, and that it was coincident with the preceding peak platform acceleration. The $NEMG_{pvc}$ and normalised acceleration data from Figure 5.22 were plotted against time in Figure 5.23, rather than platform position, to highlight this observation.

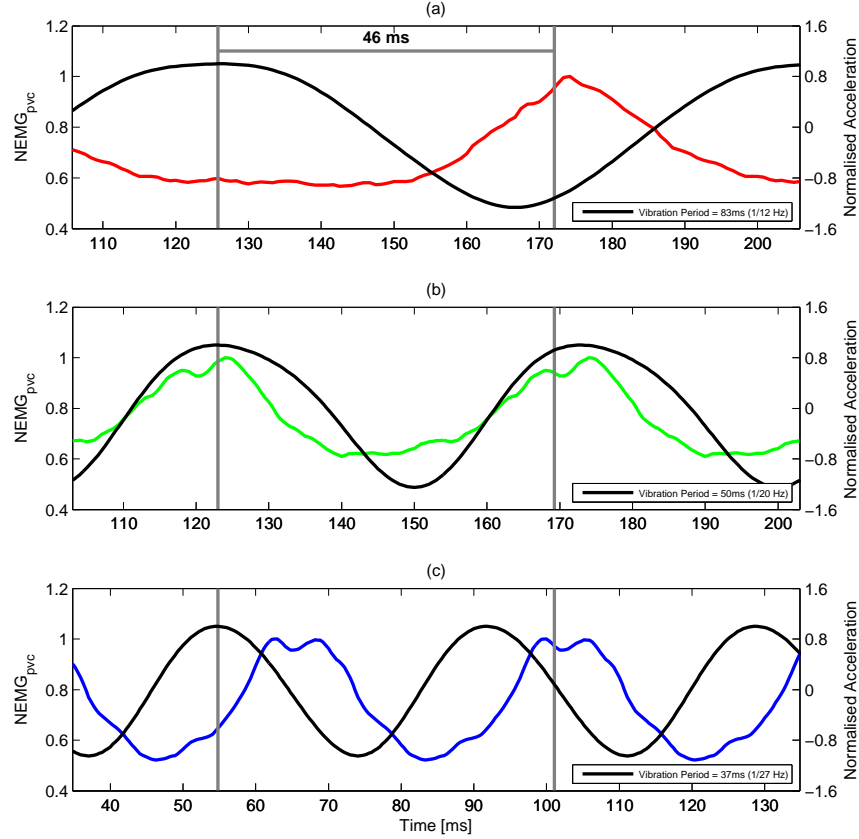


Figure 5.23: The $NEMG_{pvc}$ and normalised acceleration data from Figure 5.22 and recorded from the M-GA at the 12Hz (83ms) vibration condition (a), 20Hz (50ms) vibration condition (b) and 27Hz (37ms) vibration condition (c) were plotted against time. The normalised acceleration data sets were selected in a manner such that each of the initial peak acceleration points coincided. $NEMG_{pvc}$ was plotted against the primary y-axis and normalised acceleration was plotted against the secondary y-axis. It was determined previously that the latency between the common stimulus and the peak in $NEMG_{pvc}$ was 46ms and it is shown that this delay coincides approximately with peak acceleration. M-GA: Medial-Gastrocnemius, $NEMG_{pvc}$: Normalised EMG Per Vibration Cycle.

The process described above was repeated for the VL and TA. The results for all three muscles are shown in Figure 5.24, and the plots are equivalent to those initially presented in Figure 5.22(b).

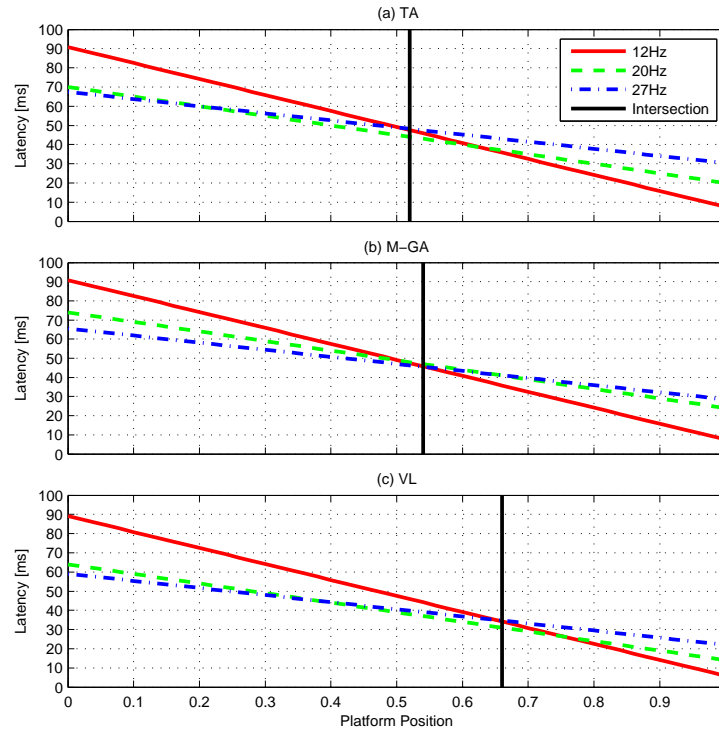


Figure 5.24: Approximation of maximum neuromuscular response initiation point. The data were grouped according to vibration frequency and muscle group. The data describe the latency (y-axis) from a potential stimulus occurring at selected points of the previous vibration cycle (x-axis) to the maximum neuromuscular response in the current vibration cycle. 0 is the beginning of the previous cycle and 1 is the end of the previous cycle (or beginning of the current cycle). The point at which the lines converge are identified by the intersection line. TA = Tibialis Anterior; M-GA = Medial Gastrocnemius; VL = Vastus Lateralis.

The intersection point was found to occur at 48 and 46% from the completion of the previous vibration cycle for the TA and M-GA respectively, and 34% for the VL. This equates to a 46.55 and 46.13ms latency between the potential stimulus and response for the TA and M-GA respectively, and 33.27ms for the VL. It is hypothesised that the shorter latency for the VL is due to mechanical delay in transmission of vibration from the foot to the leg.

5.2 Discussion

The objectives of this study were categorised as being technical and physiological in nature. The physiological objectives were: (i) to measure and quantify the magnitude of the acute, steady-state neuromuscular response of the lower limbs to WBV-pBWS and to determine the relationship between this response and vibration frequency, vibration amplitude and level of pBWS and (ii) to investigate the temporal relationship between neuromuscular activity and the position of the WBV platform. The technical objectives were: (i) to quantify the transmission of vibration from the WBV platform to the lower limbs, (ii) to estimate the proportion of vibration artefact in each EMG and then assess the suitability of active and passive EMG recording electrodes for WBV applications and (iii) to determine the optimum off-line digital filtering processes for EMG acquired during WBV and then to optimise the outlier rejection procedure. These technical and physiological objectives are discussed separately in the following sections.

5.2.1 Physiological

The physiological objectives were addressed using the EMG data with supporting data from the acceleration measurements.

EMG Amplitude Response

Every EMG was initially filtered offline, outliers were removed and then discretised according to the different test epochs: pre-Vibration, Vibration and post-Vibration. The discretised EMG were pooled according to epoch and test number (Test 1 or Test 2) and a statistical analysis was carried out which confirmed that the vibration caused a significant increase in the amplitude of the EMG and that there was no significant difference between the data recorded during the first and second testing sessions.

The magnitude of the increase in neuromuscular activity was quantified in two ways: ΔEMG (relative change in neuromuscular activity from baseline) and EMG_N (neuromuscular activity normalised to maximum (MVC)). Determining each of these parameters was important because it was previously hypothesised that the magnitude of the response to WBV may be dependent upon the state of the muscle prior to vibration. The initial condition of each muscle group was determined by measuring baseline activity prior to vibration and comparing for the different muscle groups. It was confirmed that neuromuscular activity prior to the application of vibration was different for each muscle group and was relatively large for the M-GA and VL when compared to the TA and BF. Therefore, simply comparing absolute neuromuscular activity between the different muscle groups for a given test condition was not appropriate because they were not all starting from the same baseline condition. The magnitude of neuromuscular activity prior to vibration is somewhat irrelevant to the ΔEMG value because it is the relative change that is being determined, it was therefore deemed to be useful for assessing common trends in the responses of the different muscles. However, recording EMG_N remained important also as it was used to confirm if any change in activity were clinically relevant. For example, if baseline activity was small to begin with then any small increase in neuromuscular activity would result in a large ΔEMG being calculated, however since the actual increase in activity was small originally then it may not be clinically

beneficial.

ΔEMG and EMG_N were pooled for the Vibration epoch only and grouped according to the four main factors under investigation in this study: vibration frequency, vibration amplitude, muscle group and level of pBWS. The magnitude of the neuromuscular response was significantly different for each of these grouping factors. There was a decreasing trend in the magnitude of neuromuscular activity with increasing levels of pBWS whereby ΔEMG was typically 9% at 100% pBWS and 35% and 0% pBWS. This supported the hypothesis that the level of contraction in a muscle prior to vibration affected the magnitude of the response during vibration.

Increasing vibration frequency and vibration amplitude each resulted in a statistically significant increase in neuromuscular activity, where typical ΔEMG values were between 14 - 33%. These increases were equivalent to an EMG_N of 2 - 3% MVC. It is worth noting that the average values shown here were calculated for all muscles and all levels of pBWS resulting in a small overall average being reported. Results presented later show that the changes in activity were considerably smaller when a high level of pBWS was provided compared to when a low level of pBWS was provided.

There was a significant difference between the magnitudes of the neuromuscular responses of the different muscle groups during the Vibration epoch. The largest changes occurred in the M-GA and VL, where typical ΔEMG values were 49% and 20%. These changes were equivalent to a contraction of 5% and 10% MVC respectively. The changes in the TA and BF were smaller: 20% and 15% respectively, equating to approximately 2% MVC.

These results confirmed that WBV did indeed elicit an increase in neuromuscular activity and indicate that the effect of WBV on the different muscle groups was different. This is not surprising because the acceleration data indicated that the dose of vibration received by the thigh muscles was different to that received by the shank muscles. It was also noted that the total magnitude of muscle activity for each muscle during the 0.0g condition was different also. For example, at 0% pBWS the magnitudes of the contraction for the TA, M-GA, VL, and BF respectively were 0.01%, 0.04%, 0.14%, and 0.02% MVC. These results confirmed that an intermuscular comparison of the Vibration epoch results was not appropriate in this instance.

This was supported by investigating the relationship between pre-tension and the response to WBV, i.e. by comparing EMG_N for the pre-Vibration and Vibration epoch. Figure 5.14 showed this relationship for each muscle group respectively. A best fit trend line was overlaid the scatter plot to indicate the general relationship between the initial level of muscle activity and that which occurred during the Vibration epoch. There was a positive and statistically significant increase in muscle activity for all vibration conditions confirming that there was an increased neuromuscular response to WBV. However, the type of response for each muscle was different as indicated by the slope of the trend line. It was noted that the slope for the shank muscles was an order of magnitude larger than that of that for the thigh muscles. It was therefore concluded that it would be inappropriate to group the responses of all the muscles together and that they should be assessed on an individual basis.

EMG_N and ΔEMG were assessed for each individual muscle, grouped according to level of pBWS and plotted in Figures 5.15 - 5.18. The same general trend was observed for all muscles and the salient results are summarised in Table 5.11 below. These results supported those originally described in Figure 5.14 which showed a steeper slope translated into a larger change in neuromuscular activity relative to baseline.

Table 5.11: Range of typical EMG_N and ΔEMG values for each muscle. EMG_N and ΔEMG data were grouped according to level of pBWS and platform acceleration for all participants and tests. The median of each set of grouped data was found. The maximum and minimum median values presented are representative of the typical range of values found. pBWS: partial Body Weight Support; TA: Tibialis Anterior; M-GA: Medial Gastrocnemius; VL: Vastus Lateralis; BF: Biceps Femoris; EMG: Electromyogram; MVC: Maximum Voluntary Contraction; EMG_N : EMG amplitude normalised to MVC; ΔEMG : Relative change in EMG between pre-Vibration and Vibration normalised to the Vibration value.

pBWS [%]	Muscle	EMG_N [%]			ΔEMG [%]		
0	TA	1.1	-	2.0	21.8	-	55.5
	M-GA	5.9	-	18.9	27.2	-	71.7
	VL	17.7	-	26.5	13.4	-	25.0
	BF	2.5	-	8.1	7.6	-	68.3
50	TA	0.7	-	1.2	6.9	-	29.7
	M-GA	3.6	-	12.6	22.6	-	73.7
	VL	9.4	-	10.9	15.5	-	24.4
	BF	1.8	-	2.0	3.2	-	35.2
100	TA	0.7	-	0.9	4.5	-	22.7
	M-GA	1.5	-	7.5	5.6	-	67.5
	VL	1.6	-	1.7	20.3	-	26.5
	BF	1.3	-	1.5	3.7	-	8.6

In summary the results showed that: (i) the most active to least active muscles during the Vibration epoch were VL, M-GA, BF and TA respectively. This order was irrespective level of pBWS provided; (ii) the largest to smallest change in neuromuscular activity relative to the pre-Vibration epoch was M-GA, TA, VL and BF and this was consistent with the slope values presented in Figure 5.14; and (iii) neuromuscular activity decreased with increasing levels pBWS and ΔEMG decreased accordingly as a consequence.

The main factors under investigation in this study were vibration frequency, vibration amplitude, muscle group and level of pBWS. The change in neuromuscular response during the Vibration epoch could not be attributed to any single one of these factors but was rather a combined response to all of them. However, general trends were observed which were consistent across all muscle groups. It was confirmed that the initial conditions for the different muscle groups were different and this had a significant effect upon the type of response from each muscle.

It was concluded that the difference in response was primarily due to the relative proximity of the muscle group to the vibration platform. Better vibration transmission resulted in a

larger relative change in neuromuscular activity when compared to baseline. For example, the change in activity of the shank muscles was relatively large compared to the change in response of the thigh muscles, whereby the change in activity of the shank muscles was 2.3:1 compared to 1.3:1 for the thigh muscles. Despite this however, the absolute levels of neuromuscular activity of the VL muscle group was highest, most likely because the level of pre-contraction in the VL was largest in order to facilitate standing in a shallow squat positing. It was therefore concluded that though the relative change in activity was larger in the M-GA the potential therapeutic benefit of WBV would be larger in the VL. Overall however, neuromuscular activity during WBV applied in this manner remains small with little potential for hypertrophy or an increase in muscle performance.

EMG Temporal Response

The amplitude response analysis above was concerned with the steady-state neuromuscular response to WBV only. The analysis was predicated on a RMS calculation which is fundamentally an averaging technique. The RMS averages out most of the variability in the EMG within the range over which the RMS calculation is carried out. In this study the RMS was calculated over a 0.5s moving window which was considerably larger than a single vibration period. A qualitative review of the time series EMG data was therefore carried out and characteristic peaks in the EMG were identified. The peaks were not be considered to be artefacts because they were removed from the data prior to this review. Such peaks were characterised previously by Ritzmann *et al.* [88] as a stretch reflex which was locked in phase with the period of vibration. The peaks were shown to occur once per vibration cycle and the latency to the maximum of the peak was the same as that of a short-latency reflex. They therefore concluded that the neuromuscular response to WBV was based on the stretch reflex.

A similar analysis was carried out using this data set. However, general trends were investigated here rather than analysing each individual EMG on a case by case basis. This was done by using the acceleration data to discretise every EMG to a single vibration period. The average over all periods was then calculated. In this manner genuine and regular increases in the EMG amplitude which occurred at a specific instance during a vibration period should be apparent and small, random changes in the EMG amplitude should be minimised. The outcome of the analysis was a single characteristic 'EMG pattern' that was of one vibration period in duration. This analysis was thus used to assess two alternative hypotheses: (i) was the increase in neuromuscular activity random during WBV? or (ii) was there a particular instance during a WBV cycle that elicited a specific increase in neuromuscular activity?

Every EMG that was recorded in this study was analysed in this manner. The EMG patterns were initially grouped and averaged according to the four main factors under investigation: vibration frequency, vibration amplitude, level of pBWS and muscle group. No characteristic pattern was identified for the pre-Vibration or post-Vibration epoch as expected. This is typical of a CMAP recorded by surface EMG electrodes from a muscle under normal contraction (e.g. when there is no external stimulus such as a patellar tendon tap or electrical stimulation of the α -motoneuron). In contrast to this, a specific and discernible increase in the amplitude of the EMG pattern was found during the vibration epoch. The increase in amplitude was particularly discernible for the data grouped according to vibration frequency

and supports those results found by Ritzmann *et al.* [88].

The peak response of the shank muscles was found to occur approximately 7, 24 and 29ms after the initiation of the vibration cycle for the 12, 20 and 27Hz vibration conditions respectively. If it is initially assumed that the occurrence of this peak was the result of an elicited response, and in the knowledge that latency of a monosynaptic reflex for these muscles is approximately 30ms [55], it is therefore unlikely that the stimulus occurred within the present vibration period and was therefore more likely to have occurred within the previous vibration period. Further analysis confirmed this hypothesis.

The analysis indicated that the neuromuscular response during WBV was not random, but rather there was a specific instance during which the amplitude of the neuromuscular response increased rapidly. The cause for this increase is inconclusive, but the data suggests that there was a point during the WBV cycle that acted as a trigger which subsequently elicited a maximal neuromuscular response. If it is assumed that this trigger was common for all test conditions and that it occurred at the same instance irrespective vibration frequency, vibration amplitude or level of pBWS, then these data show that the latency between the trigger and maximal response was approximately 46ms for the TA and M-GA respectively, and 33ms for the VL. This corresponds to approximately 50% completion of the previous vibration cycle for the shank muscles, which was one of the two points at which maximum platform acceleration occurred. The estimated trigger for the VL occurred at approximately 68% of the previous vibration cycle, which was also a point of maximum acceleration. Note that the point of maximum acceleration of the VL lagged that of the platform and foot (Figure 5.4) and maximum acceleration of the VL actually occurred at 30% and 70% completion of a single vibration cycle. It was thus concluded that were there to be a specific stimulus during a single WBV cycle that elicits a short-latency reflex type of neuromuscular response, then peak platform acceleration is likely to be the trigger.

In summary, a relationship between the neuromuscular response to WBV and a single cycle of vibration was identified in this study. However, in contrast to the findings by Ritzmann *et al.* [88], the ‘phase-locking’ of the EMG to the cycle of vibration was not as distinct in these results. The relationship identified in this study was a peak that consistently appeared in the EMG in phase with the vibration frequency, however the peak is described as being more broadband in nature, rather than a distinct peak that could be described as having a fixed latency analogous to a stretch reflex. It is hypothesised that this is most likely due to the fact that this analysis pools the response of all ten participants rather than assessing each EMG on an individual basis. In addition, this analysis assumed common mechanical delay between the vibration platform and the elicited neuromuscular response. In practice, the participants were of differing height and body composition, each of which may have affected the transmission of vibration. Furthermore, in all cases the latency of the response was larger than that which would be expected for a short latency reflex. It was therefore concluded that the maximal neuromuscular response was elicited by the preceding instance of maximum platform acceleration, and the lag between peak acceleration and peak response corresponded to the time taken for the muscle to respond to the stimulus.

5.2.2 Technical

The technical objectives were predominantly addressed using the acceleration data.

Vibration Transmission

Acceleration measured during vibration comprises two main characteristic components: frequency and amplitude. These individual components were derived from the acceleration signals measured in this study in order to confirm the vibration parameters being delivered during WBV and to quantify the dose of vibration received by different segments of the body during WBV.

A larger weighting was initially given to acceleration and frequency data rather than amplitude when interpreting these results, because of the increased likelihood compounding any quantization error in the raw acceleration data during integration. The likelihood of this occurring was minimised by band pass filtering the acceleration data within the frequency range of interest (10 - 30Hz) therefore eliminating high and low frequency noise components, however, the complete elimination of these error sources was never guaranteed.

Displacement was recalculated using a frequency based method to confirm the results of the integration method in an effort to improve confidence in the data. The frequency based method comprised of identifying the peak acceleration value of each vibration cycle and then averaging for the full data set. Vibration frequency of the same data set was then identified by computing the PSD and finding the peak. The only remaining unknown was vibration amplitude which was then calculated using Equation 3.1 (Chapter 3). The results from the integration and frequency based processing method were compared and found to be the same and therefore only one set of the results were presented in this thesis.

In summary, the results showed that vibration frequency was perfectly correlated between all test conditions at all measurement locations. Acceleration and displacement measurements taken directly from the WBV platform closely matched the expected values. Acceleration and displacement of the foot was well correlated but not in exact synchrony with the vibration platform. Meanwhile, an increase in acceleration and displacement of the VL was indicated for all test conditions, though it was not well correlated with that of the WBV platform.

The positive correlation between platform and foot displacement and poor correlation between platform and VL displacement was expected. The feet were in direct contact with the WBV platform and were therefore expected to closely match platform movement. The feet were simply moved across the vibration platform to achieve the different vibration amplitudes required. The corresponding movement of the VL was relatively small by comparison and was consequently unaffected by foot movement and therefore vibration amplitude. Furthermore, the VL was indirectly linked to the WBV platform via the feet and lower leg. It was therefore expected that foot placement would most likely influence foot displacement only. This observation was corroborated by the platform, foot and VL trajectories which showed that the foot and platform trajectories are well correlated but lagged by the VL trajectory. Despite this, vibration frequency was unaffected by damping and was the same at all measurement

locations for a given test condition.

A 1mm offset between platform and foot displacement was found. For example, a 1mm displacement of the vibration platform was translated to a 2mm displacement of the foot. This occurred because it was not possible to completely align the entire foot with the exact point of 1mm vertical displacement of the WBV platform. In practice the inside of the foot would have been vibrating at a smaller amplitude than this and the outside of the foot at a larger amplitude. There was a decreasing trend in the measured displacement of the foot with increasing vibration frequency also. This is likely due to the kinematics of lower limb movement during high frequency vibration. As the foot is flexible rather than perfectly rigid, it cannot match the movement of the platform dynamically at high frequencies.

By comparison the displacement of the VL was between 1 - 2mm regardless of the test condition. It was therefore concluded that displacement, and therefore acceleration, were being attenuated as the vibration was being transmitted upwards from the vibration platform. This was probably due to the uncoupling of the thigh section of the lower limbs and the vibration platform and the absorption of some of the vibration energy by the lower part of the leg.

The transmission of vibration to the body from the platform was expected to occur via the feet and skeleton. Furthermore, it was hypothesised that the propagation of vibration throughout the body was via the skeleton only. A review of the acceleration data indicated that vibration was being damped as it was transmitted upwards. This was indicated by a decrease in the absolute values of acceleration recorded from the platform, foot and thigh respectively. It was hypothesised that damping was caused by the soft tissues of the body which interconnect the skeletal segments and provide some shock-absorbing capacity. In particular, the viscoelastic properties of cartilage acts to absorb the impact energy and therefore minimise the potential for damage thus preserving the joint [180]. In addition, a mechanical model has described how elastic properties of the muscles and tendons act to absorb vibration energy and store it for one half of a vibration cycle, and then transfer the vibration back to the platform for the second half of the vibration cycle [82]. Thus, minor inefficiencies in tendons will cause some of the vibration energy to be dissipated as heat, therefore reducing the transmission of the amplitude of vibration through each subsequent joint. Furthermore, muscles themselves are thought to play a significant role in the reduction of vibration transmission. Wakeling *et al.* [78] initially showed that the lower limb resonates between 10 - 50Hz depending on joint angle and contraction. They established that this resonant frequency can be modulated by increasing or decreasing muscle activity and therefore muscle tension. They described that common walking and running conditions result in a heel strike frequency that easily falls within this range. In a subsequent study they put forward a theory that for a given input at or near the limb resonant frequency, fast-twitch, Type II muscle fibers act to change the tension within a muscle and therefore move the resonant frequency of the limb away from the input frequency, thus attenuating vibration amplitude and therefore reducing the likelihood of damage [84]. In this manner the the amplitude of the vibration is damped as it is transmitted longitudinally through the body. Damping of vibration was therefore expected and indeed did occur during these experiments. Analysis indicated that damping did not

affect vibration frequency. It was therefore concluded that the reduction in acceleration as it was transmitted throughout the legs was a direct result of a reduction in vibration amplitude only. In all likelihood, this reduction was due to the absorption of vibration energy by the shank muscles.

Analysis of the vibration amplitude results from each recording location supported this conclusion. Displacement recorded from the vibration platform was generally well correlated to that set for the test, with a slight overestimation at 12Hz only. However, displacements recorded from the foot and thigh were not well correlated. Displacement of the foot increased, but not in proportion with the displacement set for the test, and displacement of the thigh increased during vibration but was typically unrelated to that of the WBV platform.

It was not possible to empirically determine the magnitude of damping which occurred at each lower limb skeletal segment using this data set. Acceleration was recorded using sensors affixed to the outside of the body in this study. It was therefore not possible to differentiate between damping that occurred at each skeletal segment, and that which occurred between a skeletal segment and the local acceleration sensor. This led to the conclusion that this recording setup may be used infer general trends of damping only but may not be used to quantify it. The transmission of vibration was generally reduced as proximity to the source of vibration increased.

Three principal implications of damping were identified, the first being that damping implies the absorption of the vibration energy at some point lower down in the legs. This absorption of energy most likely occurred in the muscles which contracted in order to counteract the vibration. Secondly, any therapeutic effect attributable to vibration may be reduced with decreasing proximity to the WBV platform. Finally, a reduction in the proportion of vibration artefact present in EMG recorded at an increased distance from the WBV platform was expected because movement of the electrode appeared to diminish therefore reducing the potential for the occurrence of movement artefact.

In summary, it was concluded that the relative dose of vibration received by a muscle from the WBV platform could be inferred from the acceleration data. The attenuation of acceleration implies a reduction in vibration amplitude and not vibration frequency. This form of damping acted to reduce the dose of vibration received by those muscle furthest from the vibration platform. It was therefore hypothesised that any change in neuromuscular activity during vibration would be smallest in those muscles that were furthest from the platform and largest in those muscles that were closest. Finally, these results indicated that vibration artefact would only be present at the fundamental frequency of vibration and its harmonics, therefore simplifying the process of removing artefact from the EMG.

Vibration Artefact

The frequency of vibration was found to induce some error into the recorded EMG in the form of artefact. The artefact acted to change the shape of the EMG, resulting in an increase in its amplitude. It was manifested as a series of 'spikes' in the PSD of the EMG which occurred specifically at the frequency of vibration and multiples of its harmonics. This error

was determined to be a limiting factor in the type of electrode that could be used to record EMG during WBV, and it was concluded that only active EMG electrodes were appropriate for such applications. This was in contrast to others who suggested that these spikes were non-significant [126] or were manifestations of stretch reflexes rather than artifact [88].

Relatively few studies have quantified the proportion of artefact contained in an EMG recorded during WBV. Earlier work has primarily been carried out by Fratini *et al.* [126], and the significantly larger quantities of artefact identified in this study was determined to be a consequence of the failure of previous work to consider that EMG electrodes placed on the patella and then electrically isolated from the skin will only detect artefact due to vibration of the cable, and not that which occurs due to movement of the electrode itself. The conclusion of this work was that artefact content was significant, and that the elimination of it from the EMG was relatively trivial due to its presence at known and specific frequencies.

The typical frequencies of interest in an EMG are between 10 and 500Hz. As a result the standard ISEK guidelines recommend band pass filtering the EMG within this range. These guidelines were adhered to in the first iteration of the analysis carried out for this study but with a high pass filter cut-off frequency of 30Hz rather than 10Hz. The higher cut-off frequency was selected because the frequencies of vibration applied in this study were between 12 and 27Hz. A large proportion of interference was therefore expected to occur within this frequency band. Qualitative checks of the EMG were carried out next, whereby a random selection of the Time Series Data (TSD) sets and frequency plots were checked for abnormal characteristics. Sharp increases in signal power were found at harmonics of the vibration frequency in all cases. This phenomenon was identified previously by others who concluded that the spikes were artefacts which occurred during impact between the legs and vibration platform [85]. A second iteration of analysis of the EMG was therefore carried out to quantify the artefact specifically.

The quantity of artefact was measured as a proportion of total EMG power to determine how best to remove it. It was most likely manifested in the EMG by the relative movement between the recording electrodes and the underlying muscle. However, relative movement between the recording electrodes themselves and movement of the EMG cables may have led to the presence of artefact also. It was concluded that an overestimation of the EMG amplitude would occur if artefact was not removed first, resulting in a considerable overestimation of the neuromuscular response being attributed to WBV.

Artefact was detected in every EMG recorded in this study. The total magnitude of vibration artefact in each EMG was quantified in order to inform the type of EMG electrode that should be used and to determine how much off-line filtering should be applied. It was ultimately concluded that artefact should be removed using fourth-order stop-band filters implemented specifically at the frequency of vibration and its harmonics. The principal limitation of this approach was that genuine neuromuscular activity which coincidentally occurred at the frequencies which fell within the stop-band ranges were eliminated from the EMG also. An underestimation of the neuromuscular response being attributed to WBV was therefore likely.

Analysis indicated that the minimum quantity of artefact present in an EMG was 7.6% and 4.2% of the total EMG signal power when recorded using passive and active electrodes respectively. However in some isolated instances, vibration artefact accounted for 95.8% and 91.4% of the total EMG signal power for the passive and active electrodes respectively. In general however the mean quantity of artefact was 42.1% of EMG power when recorded using passive electrodes, and 24.1% when recorded using active electrodes. It was concluded that passive electrodes were inadequate for WBV applications based on this evidence alone.

Several sources of vibration artefact were identified previously and included movement of the EMG electrode cable, movement of the bi-polar electrodes with respect to one another and movement of the recording electrodes with respect to the muscle underneath. Vibration of the electrode can potentially introduce noise into the acquired signal by creating an unstable potential differences between the individual cores contained within the cable. This may be minimised by twisting the individual cores together however it is generally preferable to carry out pre-amplification of the signal at the recording site prior to transmission along the cable. This acts to increase the signal-to-noise ratio and makes any noise generated by the movement of the cable negligible. A small signal-to-noise ratio was most likely the cause of the additional artefact present in the EMG recorded by the passive electrodes when compared to those recorded using the active ones. Pre-amplification was carried out at source by the active electrodes, whereas it was carried out out after transmission along the sensor cable for the passive electrodes.

The active electrodes had an added benefit whereby the bi-polar sensing elements of the electrode were enclosed within a robust housing which eliminated the potential for relative movement between them. By process of elimination it was thus determined that the relative movement of the active electrode with respect to the muscle belly underneath was the most probable source of artefact in the EMG. This was in contrast to the passive electrodes whose bipolar sensing elements were affixed to the skin separately. Relative movement of the passive bipolar electrodes with respect to one another was possible, in addition to the relative movement of each electrode with respect to the muscle belly.

The power of the first 15 harmonics of vibration were calculated as a percentage of total EMG power. High power artefact at the frequency of vibration was not found because a large proportion of this frequency was removed online by the recording hardware. The second and third harmonics of the vibration frequency did not fall within the range of the hardware high pass filters and were not removed. These frequencies thus contributed substantially to the quantity of artefact in the EMG. There was an exponential decline in quantity of artefact at the other harmonic frequencies thereafter. A statistical analysis was carried out to determine quantitatively the effect of each harmonic frequency on the amplitude of the EMG.

A statistically significant increase in the amplitude of the EMG was found at each of the first 10 harmonic frequencies when using active electrodes, but the effect size of each increase was medium to large ($ES \geq 0.1$) for the first 5 harmonics only. The results for the passive electrodes were broadly similar, where the first 9 harmonic frequencies resulted in a significant increase in the amplitude of the EMG, and the effect size of each increase was medium to large for

the first 6 harmonics only. These statistical results are summarised in Table 5.12.

Table 5.12: Summary of relevant statistical results from analysis of harmonics. n = harmonic number; $P_{total} = \sum_{i=1}^n h_i$, and is a percentage of total EMG power; Effect Size (ES) is calculated between h_n and h_{n-1} .

Active			Passive		
Harmonic [n]	P_{total} [%]	ES	Harmonic [n]	$\%P_{total}$ [%]	ES
6	19.60%	0.065	7	37.56%	0.073
10	22.78%	0.020	9	39.98%	0.037
15	24.11%	0.010	15	42.10%	0.010

In summary, over 80% of the vibration artefact recorded by the active electrodes occurred within the first 6 harmonics. The artefacts were readily identifiable in the frequency plots and caused a large and statistically significant increase in the amplitude of the EMG. A further 13.2% of vibration artefact occurred between the 7th and 10th harmonics, which was statistically significant but irrelevant in practice because the effect size was small. All remaining vibration artefact did not significantly affect the amplitude of the EMG and was non-distinguishable from regular neuromuscular activity in the frequency plots.

Similarly, over 89% of the vibration artefact recorded by the passive electrodes occurred within the first 7 harmonics. An additional 5.7% occurred between the 8th and 9th harmonics, and while this additional artefact caused a significant increase in the amplitude of the EMG, it was not readily differentiated from regular neuromuscular activity in the frequency plots and the effect size was small. All remaining artefact in the EMG was non-significant.

These results strongly suggest that vibration artefact resulted in an unacceptable increase in the amplitude of the EMG which would be best removed offline using stop-band filters prior to further processing. To ensure consistency in all subsequent analyses, it was concluded that the first 7 harmonics of vibration should be removed. A small but statistically significant proportion of artefact possibly remained in the EMG but the overall consequence of its inclusion was deemed to be negligible.

Having determined that the the first 7 harmonics contributed the majority of vibration artefact to the EMG, and concluded that they should be removed, the investigation shifted to which of the experimental conditions resulted in the generation of the largest amount of artefact. The experimental conditions analysed were vibration frequency, vibration amplitude and muscle group. An increase in artefact content with vibration frequency was found. The EMG recorded during 12Hz vibration typically comprised 14.2% artefact, compared to 36.1% for 27Hz vibration condition. Artefact content was broadly similar, at 24.0% on average, for all vibration amplitude conditions, but was typically higher in the EMG recorded from the shank muscles versus the thigh muscles. The TA and M-GA EMG comprised 24.5% and 34.9% artefact respectively, whereas the VL and BF EMG comprised 20.6% and 19.0% artefact respectively. All of these values were markedly higher for the passive electrodes.

These findings were in good general agreement with the vibration transmission results which

showed that the vibration dose received at the thigh (VL sensor) was considerably less than that received at shank (foot sensor). This suggested that the excitation of the upper leg was smaller than that of the lower leg during vibration. There was less movement and therefore less artefact content in the EMG recorded from upper leg as a consequence. The increased quantity of artefact with vibration frequency was most likely due to the fact that the excitation stimulus, which in this case was acceleration, was proportional to the square of vibration frequency. By contrast acceleration was linearly related to vibration amplitude. Thus, a larger increase in artefact content with vibration frequency relative to vibration amplitude would be expected.

In conclusion, these results confirmed that the EMG recorded during WBV contained an unavoidable but large proportion of artefact. Failure to remove such artefact would cause an overestimation of the EMG amplitude. Artefact was limited to specific frequency bands which nominally occurred at the frequency of vibration and its harmonics, which simplified the process for artefact removal. It was also confirmed that the performance of passive electrodes was inferior to that of active electrodes, and that the passive electrodes were inappropriate for EMG measurements carried out during WBV. Statistical analysis indicated that it was adequate to remove the first 7 harmonics of the vibration frequency only.

EMG Signal Processing

Vibration artefact was not the only source of error found in the EMG recorded for this study. Spurious increases in the amplitude of the EMG were identified during qualitative checks of randomly selected data sets. Such fluctuations could not be reasonably attributed to the neuromuscular response to WBV and were considered unrelated because the response was not repeatedly found for the same subject under the same conditions during the repeat test, or at the other vibration conditions. It is most likely that these random increases in the amplitude of the EMG occurred as a result of tripping or volitional movement. It was concluded that EMG which contained this error should be excluded as outliers because they could potentially bias the results.

The procedure for rejecting outlying data was automated to ensure consistency in application. In the first instance, the mean (\bar{x}) and the standard deviation (σ) of the EMG RMS was calculated, and high and low limits acceptable limits were set at: $\bar{x} \pm 3\sigma$. EMG that contained data outwith these limits were rejected as outliers. However, this technique was found to be inadequate. It was excessively sensitive for small magnitude tonic contractions (small magnitude EMG) and insensitive to large magnitude dynamic contractions (large magnitude EMG). A large standard deviation was typically found for large magnitude EMG because there was typically a large amount of underlying tonic activity in addition to burst like activity. There was therefore a large 'acceptable' band calculated which was relatively insensitive to volitional neuromuscular activity. Conversely, small magnitude EMG which were predominantly tonic in nature were calculated to have a small standard deviation. Very minor fluctuations in the EMG amplitude were flagged as outliers and being rejected as a result. A custom algorithm based on the BTR of the EMG was therefore developed and used instead.

Trial and error with qualitative spot checks of the rejected EMG resulted in an acceptable BTR value of 5. 162 EMG were ultimately rejected as outliers using this method. This accounted for 2.5% of all recorded EMG, 50% of which were recorded during the post-Vibration epoch which were not used in the subsequent analysis in any case. The majority of the rejected EMG were from the shank muscles. There was also an increase in the number of rejected EMG with increasing vibration frequency, increasing vibration displacement, and decreasing levels of pBWS.

BTR statistics of the accepted EMG were calculated. There was no statistically significant difference between tests, and the BTR was higher during the Vibration epoch than during the pre-Vibration epoch as expected. The BTR was largest in the M-GA and VL, and lowest in the TA and BF. It increased with vibration frequency and vibration displacement, and decreased with pBWS. These results suggest that there was an increased tendency away from a tonic contraction with increased stimulation intensity. It was therefore hypothesised that the participants found it increasingly difficult to maintain a consistent contraction and recruited larger muscle fibers to compensate. Larger muscle fibers are typically harder to moderate and fire with greater amplitude, therefore leading to the higher BTR values found during higher intensity vibration conditions.

5.3 Conclusions

An increase in the amplitude of the EMG was confirmed despite the rigorous process of eliminating artefact and other incidental increases in the amplitude of the EMG which were unrelated WBV. Clear and consistent increases in the amplitude of the EMG were identified for all test conditions and muscle groups. The response characteristics of each muscle group were different and complex, and it was not possible to attribute the increase in neuromuscular activity to any single test parameter. However, in general the magnitude of the response was indicated by the dose of acceleration received at each location. This was further supported by the vibration cycle analysis, whereby the peak neuromuscular response was apparently elicited by the preceding peak platform acceleration. In general the peak neuromuscular response was small for all test conditions, whereby the maximal neuromuscular response was recorded from the VL, and average values did not exceed 27% MVC.

These results confirmed that the neuromuscular response to WBV was being moderated by platform acceleration. An increase vibration frequency therefore resulted in a comparatively larger increase in neuromuscular activity than that elicited by vibration amplitude. This is due to the fact that acceleration is proportional to amplitude and the square of frequency. However, this relationship was generally limited to those muscles that were in close proximity to the vibration platform and breaks down for those muscles that are further away. Damping acted to significantly reduce the dose of vibration received by muscles further from the vibration platform, as a result an increase in platform acceleration had a limited effect on the thigh muscles. There was a relatively small increase in neuromuscular activity in the thigh muscles when compared to the shank muscles.

It is concluded that WBV may be applied with pBWS, but in all cases the magnitudes of the responses are relatively small when compared to maximum voluntary capacity of the muscle. The neuromuscular response may be moderated by increasing vibration amplitude or vibration frequency in any combination that increases acceleration. If a gradual increase in vibration stimulus is required then frequency should be fixed and displacement should be increased. If a rapid increase in vibration stimulus is required then both parameters should be increased together. Maximal responses are achieved when a muscle is under prior contraction, therefore the level of pBWS provided should be minimised and a standing position should be taken on the platform such that the muscles for treatment are required to contract. This was confirmed in this study whereby the standing posture adopted required the VL and M-GA to contract and each of these muscle groups exhibited the largest neuromuscular response to WBV.

The primary purpose of the acceleration measurements was to confirm the vibration parameters being applied and to infer the efficiency of vibration transmission. The principal limitation of the acceleration recording setup was that the accelerometers were affixed to the skin, whereas movement of the skeleton rather than movement of the surface of the skin was primarily of interest. However, affixing the sensors to the skin was the only practicable solution, particularly because it was to be used again in a clinical environment later. Notwithstanding this limitation, some reasonable conclusions were derived from the acceleration data. Measurements taken directly from the platform confirmed that the correct

vibration conditions were being applied during testing. Damping of the vibration did occur as it was being transmitted upwards whereby the dose of vibration being received further up the leg was smaller than that received lower down.

The main challenge of acquiring EMG data during WBV was the introduction of vibration induced movement artefact into the EMG signals. The results from this study confirmed those reported by others and showed that artefact was manifested as an increase in the amplitude of the EMG. Artefact signals were most readily identified as a series of ‘spikes’ in the frequency plots and a review of these plots in combination with the acceleration data confirmed that the vibration artefact occurred at specific multiples of the vibration frequency only. It was concluded that if EMG is to be measured during WBV then pre-amplified recording electrodes should be used. Their performance was shown to be significantly better than that of standard passive electrodes. It was still necessary to remove artefact from the EMG recorded using active electrode, though it was sufficient to remove the first seven harmonics of the vibration frequency only. The increases in the power of the EMG at higher harmonic frequencies thereafter was non-significant and the power of the harmonics at these higher frequencies was not distinguishable from the neuromuscular activity which coincidentally occurs at the same frequencies.

The main conclusions from this study to be carried forwards to the SCI investigation are as follows: (i) passive EMG recording electrodes are not suitable for WBV applications, due to the susceptibility of this type of sensor to vibration induced movement artefact, (ii) the increase in the neuromuscular response of the BF during WBV is small in comparison to the TA, M-GA, and VL under the same test conditions and should therefore not be monitored, and (iii) a small level of pBWS should be provided in order to maximise any potential neuromuscular response.

Chapter 6

WBV SCI

The findings from the initial study carried out with a neurologically intact population indicated that WBV-pBWS is feasible, and that there was an increase in neuromuscular activity which was directly associated with WBV. The magnitude of the neuromuscular response was largest at 0% pBWS and smallest at 100% pBWS. In addition, the neuromuscular response was larger in those muscles which were in closest proximity to the source of vibration, and in those muscles which were already under contraction or stretch. These results are in good agreement with other findings in the published research literature. It was also identified that: (i) passive EMG recording electrodes were not suitable for WBV applications, due to the susceptibility of this type of sensor to vibration induced movement artefact, and (ii) the increase in the neuromuscular response of the BF during WBV was small in comparison to the TA, M-GA and VL under the same test conditions. It was therefore concluded that this evidence was sufficient to merit further investigation with an SCI population, for whom such a system was intended.

Fourteen SCI participants were recruited from the Queen Elizabeth National Spinal Injuries Unit for Scotland: 5 CSCI and 9 ISCI. 9 discrete WBV conditions were tested with a level of pBWS which was appropriate to the functional capability of the participant. Acceleration and EMG measurements were recorded throughout each test and analysed offline. The acceleration measurements were in good agreement with the normative data gathered from the neurologically intact participants indicating that the vibration was being successfully transferred to the SCI subjects.

It was confirmed that WBV-pBWS is feasible with an early-stage SCI population. Increases in neuromuscular activity were found and the increases were related to the vibration. The vibration was well tolerated, there were no adverse incidents during testing and all of the vibration test conditions were successfully applied. Some instances of spasm, particularly in the CSCI participants, did occur and in these cases it was necessary to stop testing and wait for the spasm to stop before resuming testing. It was concluded however that though the relative change in neuromuscular activity from baseline was typically large, the absolute change in activity was small and unlikely to lead to muscle hypertrophy.

The EMG data contained a higher proportion of artefact than expected from the normative data, particularly those EMG recorded from the CSCI participants. A specific analysis was

carried out on one of the CSCI participants with a LMN lesion affecting the shank muscle groups, which confirmed that artefact was indeed present in the EMG. It was therefore concluded appropriate to apply rigorous offline filtering to eliminate all artefact from the EMG.

The ISCI participants consistently showed a larger increase in neuromuscular activity with increasing WBV intensity when compared to the CSCI participants. This was partly due to the fact that the ISCI participants carried out the tests with less pBWS than their CSCI counterparts, which was in agreement with the normative data which showed that the neuromuscular response at reduced levels of support were larger, and also partly due to the fact that the ISCI participants suffered muscle atrophy to a lesser degree. The higher proportion of artefact in the CSCI EMG is therefore explained by the smaller signal-to-noise ratio for this group.

Clear trends and statistically significant increases in EMG amplitude, which were consistent with WBV intensity, were found for the shank muscles of the ISCI participants. A small but non statistically significant increase in VL neuromuscular activity was found also. These findings are consistent with the normative data gathered previously and those reported in the literature. In addition, the relative change in neuromuscular activity from baseline was generally larger for the ISCI participants when compared to the neurologically intact subjects. There was insufficient data gathered in this study to identify any temporal relationship between neuromuscular activity and the position of the WBV platform.

Nonetheless, further testing after a period of training is warranted. Results from the neurologically intact subjects indicate that vibration at reduced levels of pBWS increases the neuromuscular response, therefore a gradual reduction in support may lead to a training benefit in SCI. Furthermore, the incidental episodes of spasm which occurred with the CSCI indicate that WBV is acting on a portion of the pathway which leads to spasm. Training with WBV may modulate the excitability of this pathway, and since the magnitude of the response from the CSCI participants with 100% pBWS was small, the training would not strengthen the muscle, which is mutually beneficial outcomes for the treatment of spasticity.

6.1 Results

A summary of the results presented in this section are described below:

- **Section 6.1.1 Accelerometer Analysis:** Average acceleration, amplitude and frequency were calculated for the vibration platform, foot, and thigh. The results for the SCI participants were broadly similar to those found for the neurologically intact participants. The full set of results are therefore not presented and instead a summary of the relevant findings are described.
- **Section 6.1.2 Artefact Quantification:** The total proportion of vibration artefact present in the recorded EMG was quantified. Again, the results were similar to those for the neurologically intact participants and a summary of the findings is presented instead.

- **Section 6.1.3 Case Study:** A randomly selected EMG is selected from a subject with an upper motor neuron lesion. The standard EMG filtering techniques are applied. The same data set is then reprocessed specifically removing vibration artefact. The two sets of processed data are then compared.
- **Section 6.1.4 EMG Classification:** The EMG were discretised into pre-Vibration, Vibration, and post-Vibration epochs and outlying data was rejected based on the BTR.
- **Section 6.1.5 General EMG Amplitude Response:** The magnitude of the EMG was quantified by computing and averaging the RMS of each epoch of data. The averaged values were then grouped according to test number, epoch, vibration frequency, vibration amplitude, muscle group and classification of SCI.
- **Section 6.1.7 Specific EMG Amplitude Response:** The averaged EMG_{RMS} data were grouped according to muscle group, vibration frequency, vibration amplitude and classification of SCI.
- **Section 6.1.8 EMG Vibration Cycle Analysis:** The EMG response over a single cycle of vibration was investigated.

6.1.1 Accelerometer Analysis

Average acceleration, vibration frequency and vibration amplitude of the platform, foot and VL were found for the ISCI and CSCI participants respectively. The same protocol as was used with the neurologically intact participants was applied and the results were broadly the same. The plots are therefore not repeated here but have been included in the appendix for reference.

There was typically only a small difference in response for the ISCI and CSCI participants at each recording location. The main differences were between the absolute acceleration values recorded at each location and those which were expected to occur. The recorded vibration platform accelerations were consistent, as indicated by the median values and small interquartile ranges, and were almost identical to the expected values. An increase in average acceleration recorded from the foot was found with increasing platform acceleration. However, the response was not well correlated with platform acceleration, particularly at higher vibration amplitudes and frequencies and did not typically exceed 4g. Vibration frequency measured at each location and that set for the test were identical in almost all cases. Finally, the differences in measured displacement for the ISCI and CSCI participants were small. Displacement measured directly from the WBV was typically within 1mm of that set for the test. Displacement of the foot and thigh increased to between 1 and 4mm, but the increases were not in proportion to those set for the test.

A review of the time series acceleration data again indicated that the motion was sinusoidal. Acceleration along the longitudinal axis was again found to be larger than either of the anterior-posterior or medial-lateral axes. Analysis of the individual components of the foot and VL acceleration profiles showed that any motion along the medial-lateral or anterior-posterior axes were predominantly out of phase with the platform along the medial-lateral axis but almost completely in phase in the longitudinal direction.

6.1.2 Artefact Quantification

This section presents the results of the analysis of vibration artefact in the EMG recorded before, during, and after the application of WBV. The analysis is the same as that done with the neurologically intact participants¹. The results for those with a ISCI and CSCI were analysed separately because it was likely that the total presence of artefact in the EMG recorded from the participants would be different, due to the differing levels of injury, severity of injury, and functional capability.

The trends in vibration artefact content were ultimately found to be similar to those found for the neurologically intact participants. The figures are therefore not repeated here but are presented in the appendix for reference. The statistical analysis indicated some minor differences to those found previously and are described in greater detail below.

Individual Vibration Harmonics Components

The contribution of frequency components, which were typically associated with vibration artefacts, to the total power of the EMG were less than 3.0% throughout the pre-Vibration and post-Vibration conditions, regardless of the classification of SCI or harmonic number. Group averages (median) were 1.7% and 2.2% for the ISCI and CSCI participants respectively. These results are comparable to those from the neurologically intact participants where the average proportion of artefact was 1%.

The average contribution of vibration artefact to the total power of the ISCI EMG was between 0.2 and 8.5% during the Vibration epoch depending on harmonic number. Maximum values up to 79.9% were detected at specific harmonic frequencies. There was a downward trend in the proportion of artefact in the EMG from the second harmonic onwards, noting that the first and second harmonic frequencies were normally cut-off by the acquisition hardware high pass filter. These results were broadly comparable to those found for the participants with no neurological deficit where corresponding average values were between 1 and 11% depending on harmonic number.

The average proportion of artefact contained within the EMG from the CSCI participants was between 0.1 and 12.5% during the Vibration epoch. Maximum values up to 93.2% were found for specific harmonic numbers. There was a downward trend in the proportion of artefact from the second harmonic onwards also. There was little discernible difference between the proportion of artefact content in Vibration or non-Vibration EMG from the seventh harmonic onwards.

Overall Vibration Harmonics Contribution

The overall average (median) contribution of all harmonic frequencies ($h_1 - h_{15}$) to the power of the EMG recorded during the pre- and post-Vibration epochs were between 9.1% and 7.9%

¹In practice, vibration induced artefact is not present in EMG recorded during pre-Vibration or post-Vibration. These EMG were treated in a similar manner to those recorded during Vibration in this analysis. This was done to establish a baseline against which EMG recorded during WBV could be compared.

for the ISCI and CSCI participants respectively.

Average values of 22.7% were found for the ISCI participants during vibration, and 29.6% for the CSCI participants. However, up to 94.3% of the power of the EMG was contained within the harmonic frequency search bands for the ISCI participants under certain test conditions. The highest values for the CSCI participants was similar at 97.5%. It was noted that these results were similar to those from the neurologically intact participants where highest values were 95%.

Statistical Analysis

The overall effect of vibration artefact on the SCI EMG was determined by carrying out a multi-way ANOVA as was done in Section 5.1.2. Harmonic number (h_1 - h_{15}) and test epoch (pre-Vibration, Vibration and post-Vibration) were used as the independent grouping factors. The results presented in Figure 6.1 are for the Vibration epoch only because this was the only instance during which artefact could occur in practice.

The first 8 harmonics resulted in a significant change in $\%P_{total}$, with a non-significant change thereafter for the ISCI participants, and likewise for the first 5 harmonics for the CSCI participants. The overall ES of these changes was estimated by computing Cohen's d for each consecutive pair of grouped data (Equation (4.17)), where each point presented in Figure 6.1 is the computed ES between h_n and h_{n-1} . A small ES (≤ 0.1) was defined as the threshold value.

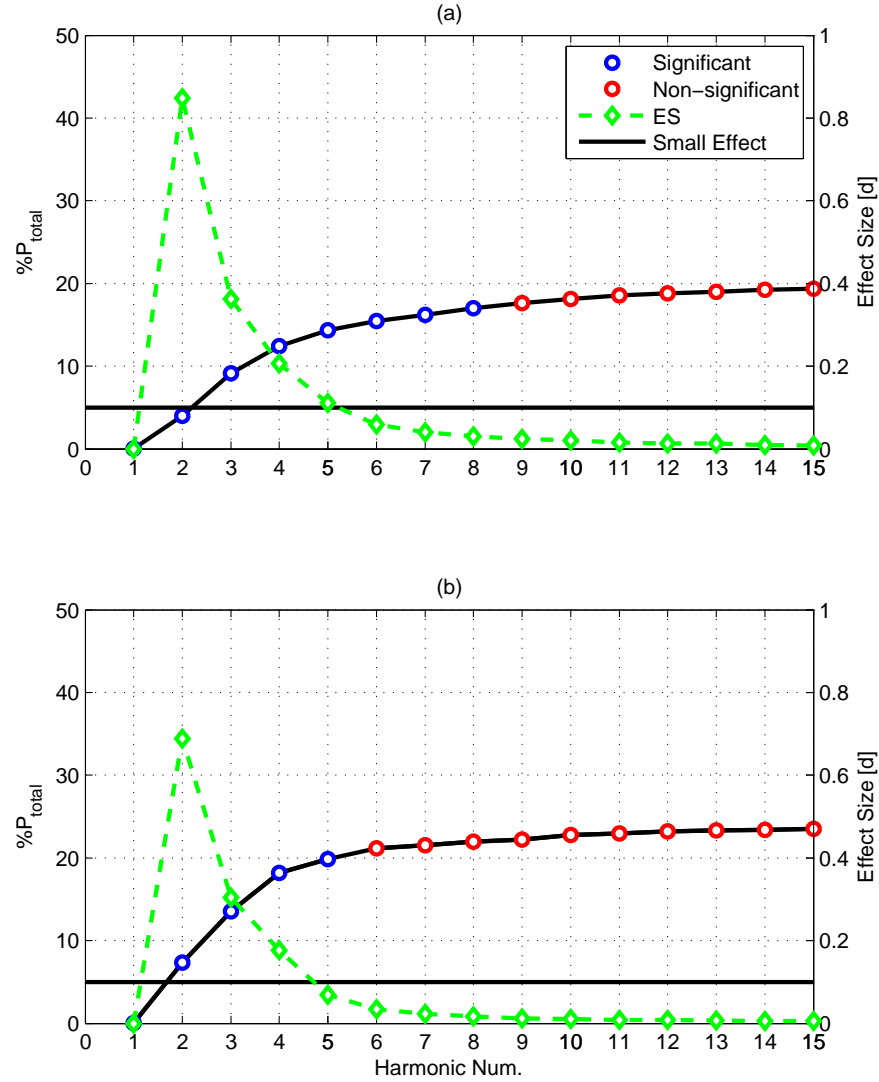


Figure 6.1: The average (median) contribution of the individual harmonics (p_{h_n}) are plotted with a solid line on the primary y-axis. The values are presented as a percentage of total EMG power for all test subjects, test conditions and EMG recorded during vibration for the ISCI (a) and CSCI (b) participants. The corresponding effect size, as estimated by Cohen's d (Equation (4.17)) are also presented as a dashed line on the secondary y-axis along with the threshold value for a small effect size of 0.1. Data that were found to be significantly different to h_{15} using an n -way ANOVA and a post-hoc multiple comparisons procedure were identified. ES: Effect size; ANOVA: Analysis of Variance.

This statistical analysis shows that h_{1-8} resulted in a significant increase in $\%P_{total}$ for the ISCI participants, and h_{1-5} for the CSCI participants. However, while statistically significant, the overall effect from h_6 onwards was small for both groups. This provides justification for the removal of h_{1-6} only. The summary below is for h_1 to h_7 in order to allow for a direct comparison with the neurologically intact participants.

Artefact Summary

The susceptibility of an EMG to movement artefact under specific experimental conditions was described using the integral of h_1 to h_7 . The integrated data were pooled according to the four main grouping factors of interest in this study: muscle group, vibration frequency, vibration amplitude and classification of SCI. The pooled data were plotted according to these grouping factors in Figure 6.2. Bartlett's test was used to assess equal variance across all grouped data sets. As equal variance was not found, the non-parametric equivalent of the ANOVA (Friedman Test) was used. The results of this statistical analysis are described in Table 6.1. Significantly different pairs were identified using a post-hoc multiple comparisons procedure with a Bonferroni correction.

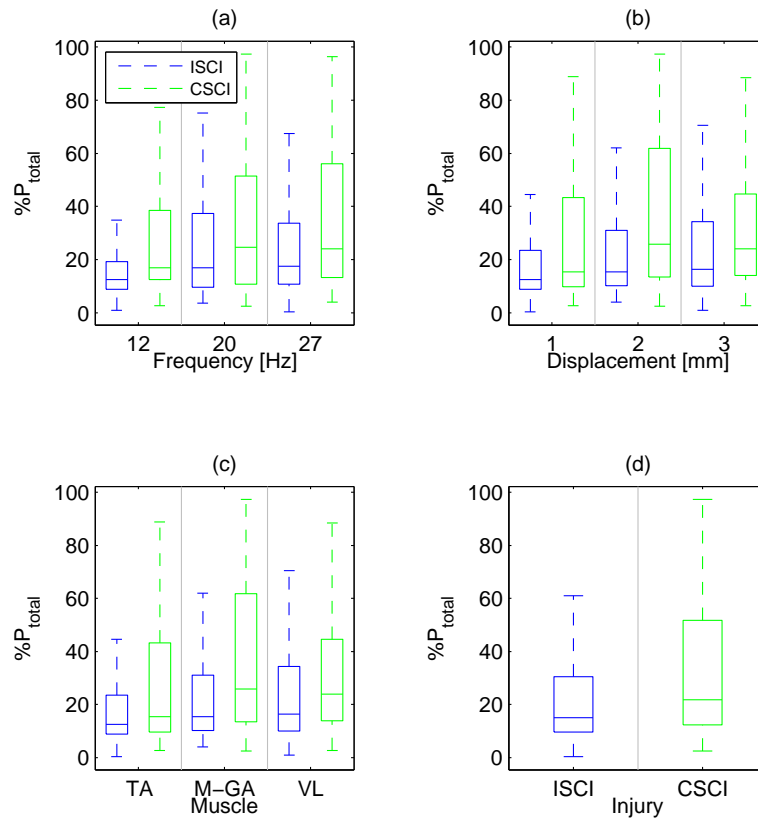


Figure 6.2: Summary of the overall contribution of the first 7 harmonics of vibration to the total power of the EMG. The contribution of the first seven harmonics of vibration were integrated for all subjects and test conditions and grouped according to vibration frequency (a), vibration amplitude (b), muscle group (c), and classification of SCI (d).

There was a larger proportion of artefact in the EMG recorded from CSCI participants than ISCI participants across all experimental conditions. Within each SCI group however, the trends were broadly similar, where: (i) there was an increase in the proportion of vibration artefact with increasing vibration frequency, and this was statistically significant for the ISCI participants; (ii) there was an increase in the proportion of vibration artefact with increasing vibration amplitude, and this was statistically significant for the CSCI participants; and (iii)

the proportion of vibration artefact was largest in the EMG recorded from the M-GA and smallest in the TA, this was in contrast to the results found for the neurologically-intact participants the proportion of artefact in the TA EMG was relatively high. The results of the statistical analysis are shown in Table 6.1.

Table 6.1: Statistical results from analysis of artefact in EMG recorded during WBV from ISCI and CSCI participants

Parameter	ISCI		CSCI	
	X ² (DF)	p	X ² (DF)	p
Frequency	19.13 (2)	≤ 0.01	3.13 (2)	= 0.21
Displacement	5.10 (2)	= 0.08	12.51 (2)	≤ 0.01
Muscle Group	5.38 (2)	= 0.07	51.53 (2)	≤ 0.01

In summary, over 74% of the vibration artefact recorded from the ISCI participants occurred within the first 5 harmonics. This vibration artefact was readily identifiable in frequency plots and resulted in a large and statistically significant increase in the amplitude of the EMG. An additional 14% of total vibration artefact occurred between the 5th and 8th harmonics. This additional artefact significantly increased the magnitude of the EMG but the effect size was small and the characteristic spikes were not easily identifiable in the frequency plots.

Similarly, over 77% of the artefact recorded from the CSCI participants occurred within the first 4 harmonics. An additional 8% of total vibration artefact occurred between the 4th and 5th harmonics, and while this additional artefact caused a significant increase in the amplitude of the EMG, it was not readily identifiable in the frequency plots and the effect size was small.

It was thus concluded that artefact content was excessively high within h_{1-7} and that it should be removed from the EMG within these frequency bands as was done with the neurologically intact participants. This conclusion was supported by the results of the following case study.

6.1.3 Case Study

The analysis of Subject S11_{SCI} provides a unique opportunity to investigate the effect of movement artefact in EMG recorded during WBV. Subject S11_{SCI} was a 19 year old male who suffered a transection of the spinal cord at the L3 segmental level resulting in a motor complete SCI. The injury damaged the motor nerves directly and was classified as a lower motor neuron lesion. It is extremely difficult to elicit a contraction in muscle innervated below this level of injury and direct electrical stimulation of the muscle cells, rather than the motoneurone, is required to cause a contraction. Typically, this may also only be achieved before extensive atrophy of the muscle cell has occurred.

Trauma at the L3 segmental level generally results in damage the tibial and peroneal nerves, which supply the TA, M-GA and BF muscle groups respectively. The femoral nerve which supplies the quadriceps muscle group is normally spared, therefore resulting in some retained functionality of the VL rather than the characteristic LMN damage associated with the the other muscles. Subject S11_{SCI} was classified as B on the AIS at the time of participation in this study, and demonstrated areflexia and a complete absence of tone in the muscles below

the level of injury. Muscle motor scores of 0 (zero) were found for the plantar flexors and long toe extensors, 4+ for the knee flexors and 5 for the knee extensors. This participant therefore had a complete absence of motor capability in the shank muscles, but near normal motor capability in the thigh muscles.

Subject S11_{SCI} participated in the same experimental protocol as every other volunteer. The EMG recorded from the TA and VL recorded during the 20Hz/2mm test condition were isolated for specific analysis in this case study, because an increased neuromuscular response from the VL was expected during vibration, meanwhile no change in TA activity was expected to occur. It was anticipated that any vibration induced artefact would therefore be easily identifiable in the TA EMG should it be present.

Figure 6.3 shows the raw EMG recorded from the TA and VL in both the time and frequency domains. The pre-Vibration and Vibration phases of the test are highlighted in the time domain plots. A $2\mu\text{V}$ increase in electrical activity was detected in the TA EMG at the onset of vibration. A larger increase between 10 and $15\mu\text{V}$ was detected in the VL EMG. The increased activity of the TA was found to occur specifically at 20Hz, and its harmonics, in the frequency domain plots. By comparison, there was a more ‘typical’ broadband increase in neuromuscular activity of the VL across the entire recorded frequency spectrum.

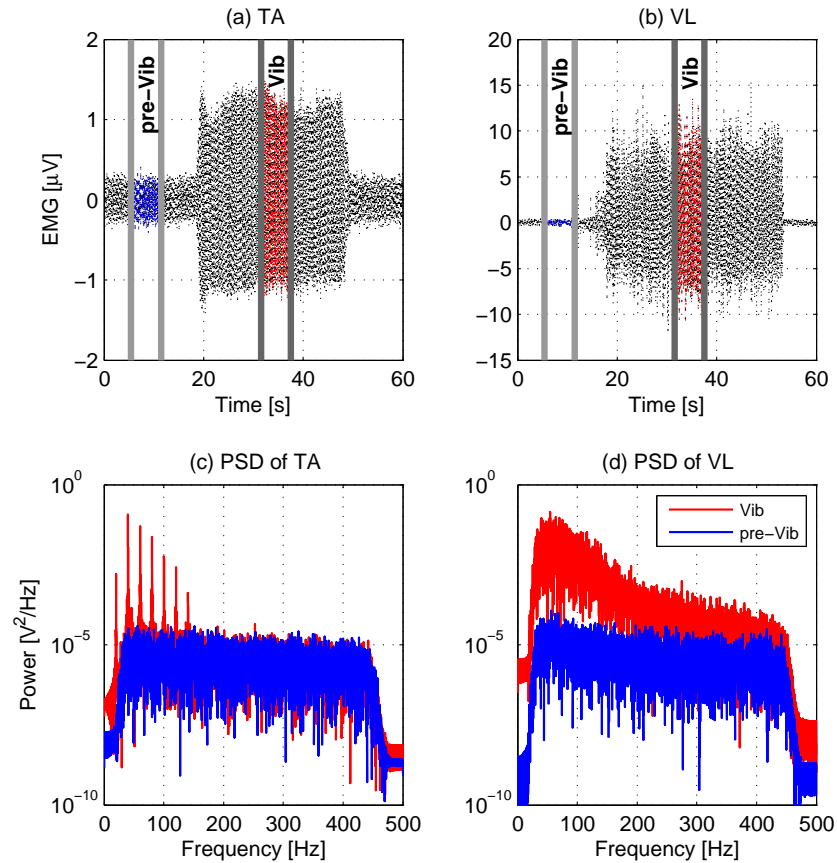


Figure 6.3: Raw electromyogram recorded from an areflexic (TA) and neurologically-intact (VL) muscle during whole body vibration. (a) Electromyogram of TA presented in the time domain. (b) Electromyogram of VL presented in the time domain. (c) Electromyogram of TA presented in the frequency domain. (d) Electromyogram of VL presented in the frequency domain. TA: Tibialis anterior. VL: Vastus lateralis. PSD: Power spectral density.

The first 7 harmonics of vibration (20, 40, 60, 80, 100, 120, and 140Hz) were removed resulting in a complete reduction in the EMG amplitude to pre-Vibration levels for TA. In contrast, removing of the same frequencies from VL, resulted in only a small decrease in the EMG amplitude.

Filtering applied in this manner left two distinct regions of increased activity in the TA EMG. These regions are highlighted in Figure 6.4(a). It was known that the vibration platform was 'ramping' up and down from the target vibration frequency during these periods from the acceleration measurements, which in this instance was 20Hz. The notch filters removed this frequency and its harmonics only, and did not remove those frequencies which occurred during ramping stages. The movement artefact which was introduced during these stages was therefore not removed from the EMG. This was not considered to be important in this analysis because the neuromuscular activity which occurred during these periods was not of interest.

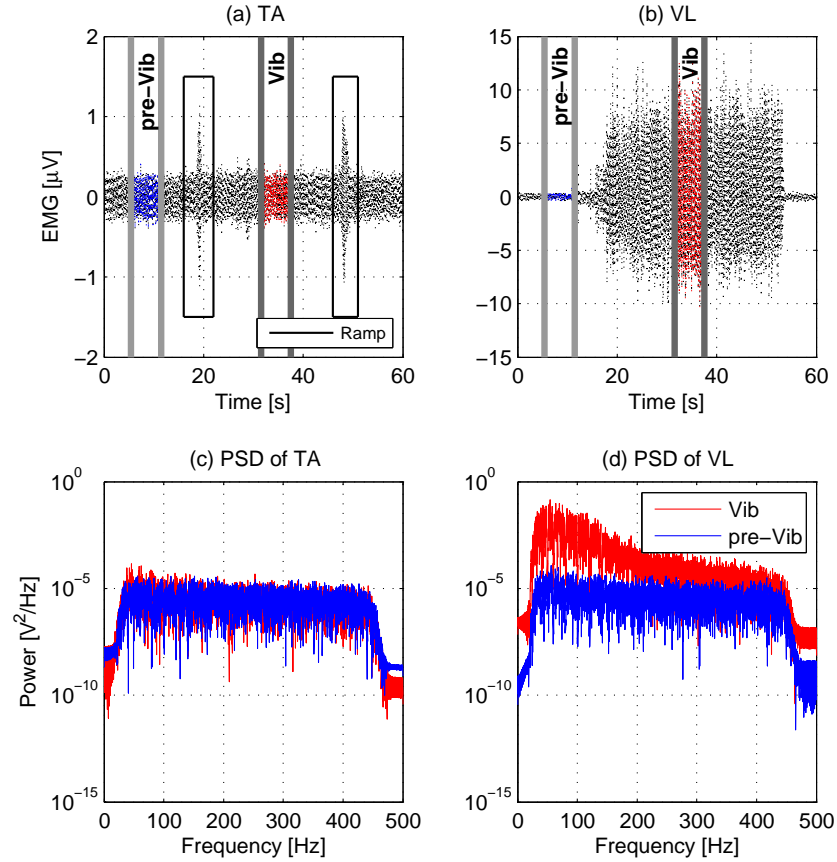


Figure 6.4: Filtered electromyogram recorded from an areflexic (TA) and neurologically-intact (VL) muscle during whole body vibration. (a) Electromyogram of TA presented in the time domain. (b) Electromyogram of VL presented in the time domain. (c) Electromyogram of TA presented in the frequency domain. (d) Electromyogram of VL presented in the frequency domain. TA: Tibialis anterior. VL: Vastus lateralis. PSD: Power spectral density.

The primary outcome measure used in this thesis to assess the magnitude of the neuromuscular response to WBV was the EMG_{RMS} . This is the most common parameter used for such analyses and is recommended by ISEK [176]. The EMG_{RMS} of the data present above was calculated using a 0.2s moving window and the results were plotted against time in Figure 6.5. The amplitude of the TA EMG was confirmed to be reduced to pre-Vibration levels for the entire Vibration epoch. The reduction in the VL EMG amplitude as a result of filtering was small by comparison.

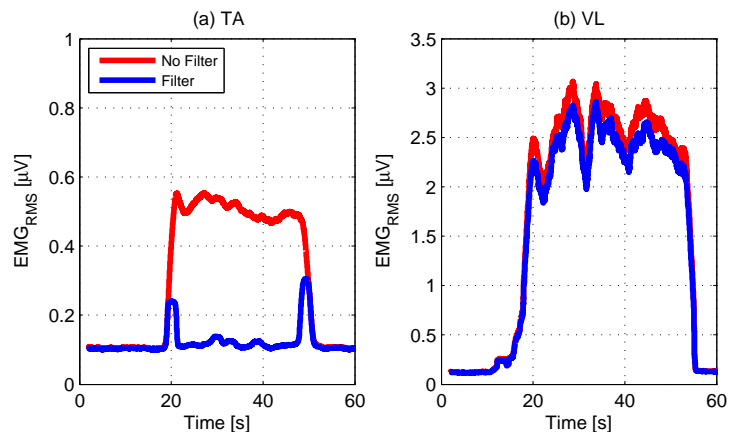


Figure 6.5: Filtered electromyogram recorded from an areflexic (TA) and neurologically-intact (VL) muscle during whole body vibration. (a) RMS of TA. (b) RMS of VL. TA: Tibialis anterior. VL: Vastus lateralis.

It was concluded from this case study that WBV resulted in a broadband excitation of the muscle, rather than simply inducing a neuromuscular response specifically at the frequency of vibration. Furthermore, the off-line stop-band filters applied in this analysis were adequate, and effectively eliminated vibration artefact from the EMG.

6.1.4 EMG Classification

A total of 4,536 unique EMG were recorded from the SCI participants in this study. Of these recorded EMG, it was those which exhibited a ‘steady-state’ response to WBV which were of principal interest. The variability of each EMG was therefore estimated by computing the BTR, which was then used to classify and reject outlying data. This section describes such outlying data and quantifies those eliminated from further analysis.

Some instances of clonic spasm or contracture occurred during testing. It is plausible that such spasm was induced by the WBV platform, but this phenomenon is not of principal interest here because such activity was not classed as steady-state, nor found to occur repeatedly for a given set of experimental conditions. Such EMG were therefore classified as outliers and ignored from further analysis.

A total of 680 (15.0%) EMG were classified as outliers and were rejected. These data are summarised in Table 6.2. There was an even spread in EMG rejected from each of the muscle groups. An increasing trend in the number of rejected EMG was identified with increasing vibration frequency and vibration amplitude. There were a larger number of EMG rejected from the ISCI participants than the CSCI participants, and the majority of EMG were rejected during the post-Vibration epoch when compared to the pre-Vibration and Vibration epochs. These trends are consistent with those from the neurologically intact participants, though the proportion of rejected EMG is higher, most likely a result of the occurrence of spasm during testing.

Table 6.2: Breakdown of rejection rates

Parameter	Composition of Rejected Values		
Muscle	TA	M-GA	VL
	35.0%	28.4%	36.6%
Frequency	12Hz	20Hz	27Hz
	26.2%	30.0%	43.8%
Displacement	1mm	2mm	3mm
	30.7%	27.6%	41.6%
Phase	pre-Vib	Vib	post-Vib
	27.2%	29.6%	43.2%
Injury	iSCI	cSCI	-
	54.9%	45.1%	-

The BTR was largest during the Vibration epoch and typical values for all epochs were between 0.25 and 1. A breakdown of the common values are shown in Figures 6.6 and 6.7.

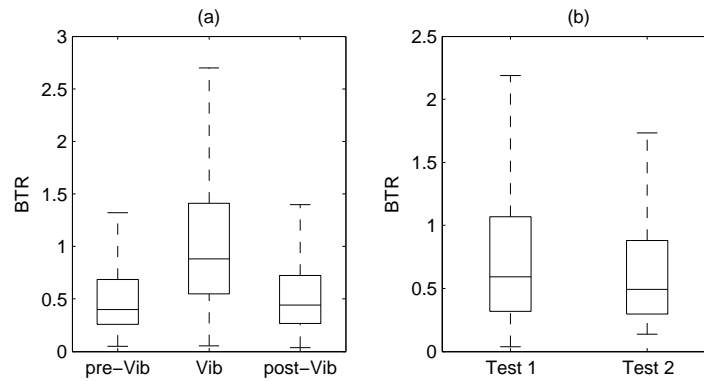


Figure 6.6: Summary of the BTR data for the EMG not rejected. The data presented are for all subjects and tests and are grouped according to epoch (a) and test number (b).

The BTRs increased with both vibration frequency and vibration amplitude, and were larger for those with an iSCI. No difference in BTR of the different muscle groups was identified. It was hypothesised that the larger BTR values found for the iSCI participants was attributable to the fact that a vibration induced response was occurring, in a similar manner that of the neurologically-intact participants. By contrast, the small BTR found for the cSCI participants was attributable to their small neuromuscular response to WBV.

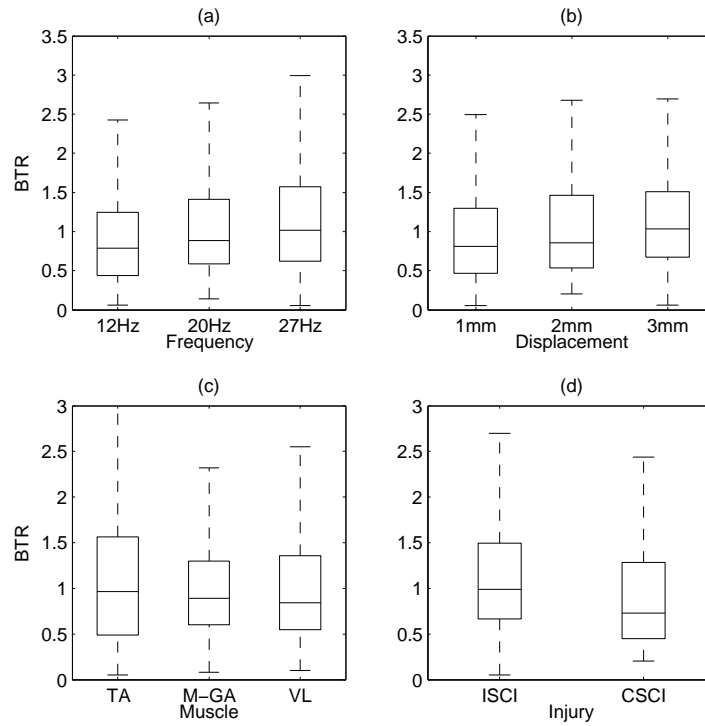


Figure 6.7: Overall summary of BTR data not rejected during the Vibration epoch. The presented data are for all subjects and testing sessions, and are grouped according to vibration frequency (a), vibration amplitude (b), muscle group (c), and classification of SCI (d). CSCI: motor-complete SCI; ISCI: motor-incomplete SCI; SCI: spinal cord injury.

A total of 3,856 recorded EMG were retained for further analysis.

6.1.5 General EMG Amplitude Response

The intention of this initial analysis was to determine the general underlying trends which described the neuromuscular response to WBV in SCI. The assessment then quantified the magnitude of this response according to the four main grouping factors which were being investigated: (i) vibration frequency, (ii) vibration amplitude, (iii) muscle group and (iv) classification of SCI.

General Results

Average EMG amplitude results are shown in Figure 6.8. The neuromuscular response was larger during Vibration when compared to the pre- and post-Vibration epochs, and this increase was determined to be statistically significant (Table 6.3). Overall, the magnitudes of the neuromuscular responses were larger during the first testing session when compared to the second.

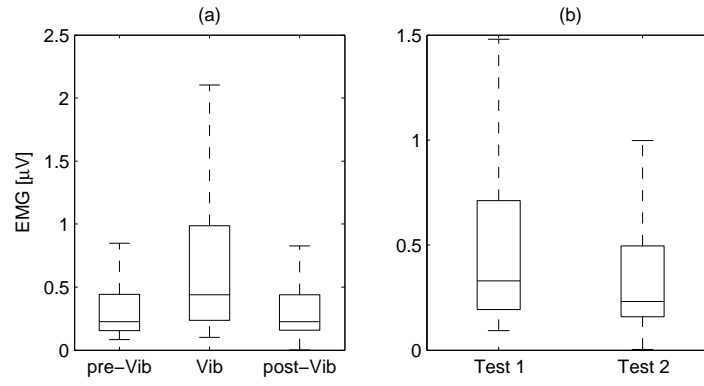


Figure 6.8: Overall summary of EMG results not rejected. Data for all subjects and test conditions are shown and were grouped according to test epoch (a) and testing session (b).

The statistical results were based on a multi-way ANOVA. The independent grouping factors were test epoch and test condition. Where significance was identified a post-hoc multiple comparisons procedure with Bonferroni correction was used to identify significantly different pairs of grouped data. The results of the statistical analysis are reported in Table 6.3.

Table 6.3: General summary of statistical results. F: ANOVA F-statistic; DF: Degrees of Freedom; p: P-value; Gu_{Gl} : Groups identified to be significantly different in post-hoc analysis; Gu: Upper group; Gl: Lower group; †: All pairs found to be significantly different at $p \leq 0.05$.

Parameter	EMG		
	F (DF)	p	Gu_{Gl}
Epoch	45.5 (2)	≤ 0.05	$Vib_{pre-Vib}; Vib_{post-Vib}$
Test	51.4 (1)	≤ 0.05	†

Vibration Results

Having confirmed that there was a significant increase in neuromuscular activity during the Vibration epoch, the analysis next investigated how the change in neuromuscular activity was related to: (i) vibration frequency, (ii) vibration amplitude, (iii) muscle group and (iv) classification of SCI. The relative changes in activity (ΔEMG) (Equation (4.12)) were calculated by comparing pre-Vibration (baseline) and Vibration EMG². In addition, the corresponding absolute values of EMG amplitude were compared. These data are presented in Figure 6.9 and results of the statistical analysis are shown in Table 6.4.

Statistical results are based on a multi-way ANOVA for the EMG recorded during the Vibration epoch only. The data were analysed for all subjects and test conditions, and the independent grouping factors were vibration frequency, vibration amplitude, muscle group and classification of SCI. A post-hoc multiple comparisons procedure with a Bonferroni

²Note that the pre-Vibration EMG were typically small when compared to those from the ISCI or neurologically intact participants. ΔEMG was therefore more sensitive to any change in neuromuscular activity by comparison. The interpretation of ΔEMG results should therefore be carried out in conjunction with the raw EMG results.

correction was used in the event that statistical significance was identified. The results of the statistical analysis are presented in Table 6.4.

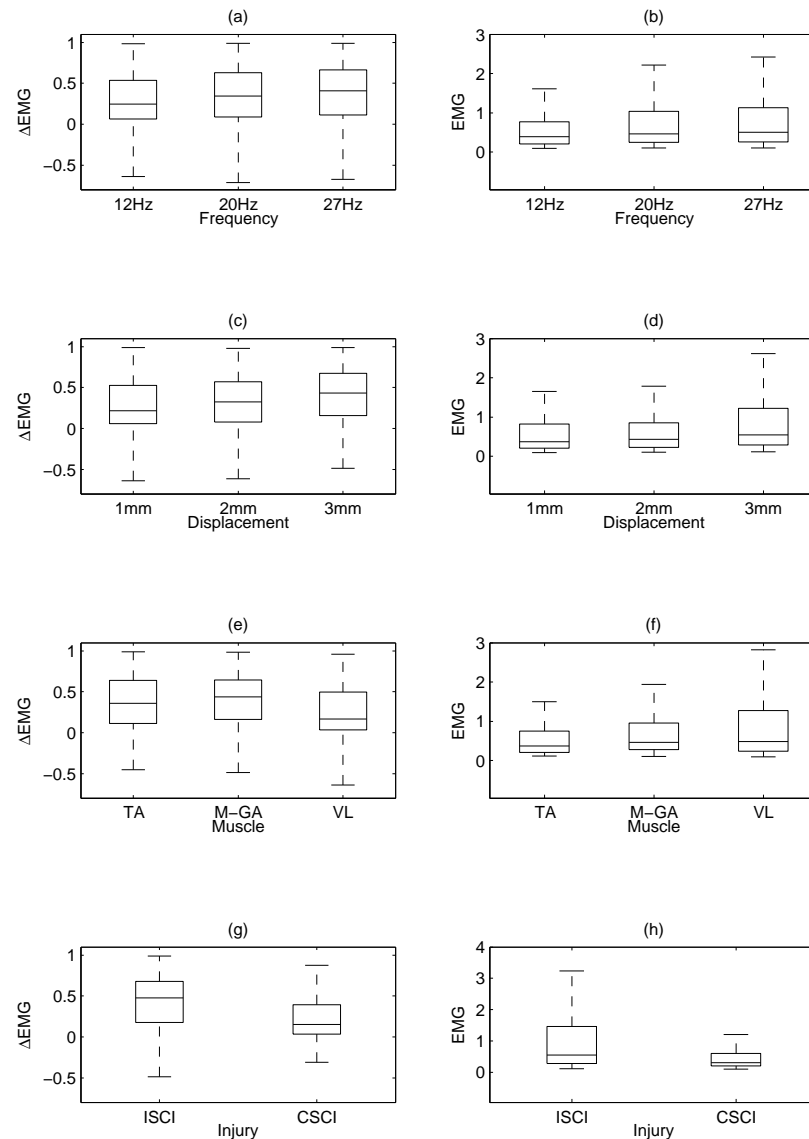


Figure 6.9: Summary of EMG amplitude response for data acquired during the vibration phase and not rejected as outliers. The data shown are the medians and interquartile ranges, and are for all subjects and test conditions. Relative changes in activity with respect to baseline are shown in (a), (c), (e) and (g) and absolute levels of activity are shown in (b), (d), (f) and (h). The data were grouped according to vibration frequency ((a) and (b)), vibration amplitude ((c) and (d)), muscle group ((e) and (f)) and classification of SCI ((g) and (h)). TA: Tibialis Anterior, M-GA: Medial Gastrocnemius, VL: Vastus Lateralis, ISCI: motor-incomplete SCI, CSCI: motor-complete SCI, SCI: spinal cord injury.

Neuromuscular activity increased with both vibration frequency and vibration amplitude. Increases were smaller for the CSCI participants when compared to the ISCI participants. Δ EMG was between 25 and 41% for the different frequency conditions, which equated to a 0.38 to 0.50 μ V change in neuromuscular activity. These changes were statistically significant, and a post-hoc analysis showed that vibration at 27Hz elicited a significantly larger increase in neuromuscular activity than both 12 and 20Hz. Similar increases were found for vibration amplitude, and the increases were significantly larger at 3mm when compared to a vibration amplitude of 1 or 2mm. Changes in vibration amplitude resulted in a range of Δ EMG values between 21 and 43%, which equated to a 0.37 to 0.54 μ V increase in EMG amplitude. Vibration caused a larger increase in activity for the ISCI when compared to the CSCI participants, where the overall Δ EMG was 48% and 15% for each sub-group respectively.

Δ EMG was similar for each of the TA and M-GA, and smallest for the VL. In absolute terms, however, the VL was the most active during vibration. Typical Δ EMG values were between 36 and 44% for the TA and M-GA, and 17% for the VL. These Δ EMG values equated to a 0.38 - 0.46 μ V increase in EMG activity. These trends are consistent with those found for the neurologically-intact participants. The Δ EMG values for the SCI and neurologically intact participants are directly comparable when grouped according to vibration frequency, vibration amplitude and muscle group. In all cases the values for the SCI participants are either the same or marginally larger than those for the neurologically intact participants.

Table 6.4: Summary of statistical analysis results. Δ EMG: Change in EMG during Vibration with respect to pre-Vibration condition; EMG: EMG recorded during vibration; F: ANOVA F-statistic; DF: Degrees of Freedom; p: P-value; Gu_{GI}: Groups identified to be significantly different in post-hoc analysis; Gu: Upper group; Gl: Lower group; †: All pairs found to be significantly different at $p \leq 0.05$.

Parameter	Δ EMG			EMG		
	F (DF)	p	Gu _{GI}	F (DF)	p	Gu _{GI}
Frequency	16.67 (2)	≤ 0.01	27 ₁₂ ; 20 ₁₂	1.923 (2)	= 0.15	-
Displacement	29.58 (2)	≤ 0.01	†	3.18 (2)	= 0.04	3 ₁
Muscle	45.75 (2)	≤ 0.01	VL _{M-GA} ; VL _{TA}	2.89 (2)	= 0.06	-
Injury	218.6 (1)	≤ 0.01	†	63.89 (1)	≤ 0.01	†

6.1.6 Case Analysis (S17)

The average TSI for the SCI participants overall, excluding Subject S17, was 171 days, and 164 days for the CSCI participants only. By comparison the TSI of Subject S17 was 5087 days. Subject S17 was a 28 year old male who suffered a traumatic spinal cord injury at T7, resulting in motor-complete paralysis below the level of lesion with some retained sensation. Subject S17 was an attendee at the Queen Elizabeth National Spinal Injuries Unit and undertook regular FES-Leg Cycle Ergometry training sessions. He met the inclusion criteria for this study and a unique opportunity was presented to compare and contrast the response of a chronic SCI participant against the responses from early-stage SCI participants.

The average (mean) EMG and Δ EMG responses of the TA, M-GA and VL were calculated and isolated for each CSCI participant and plotted in Figures 6.10 - 6.12 below. The same values were calculated for Subject S17 and overlaid the same plots.

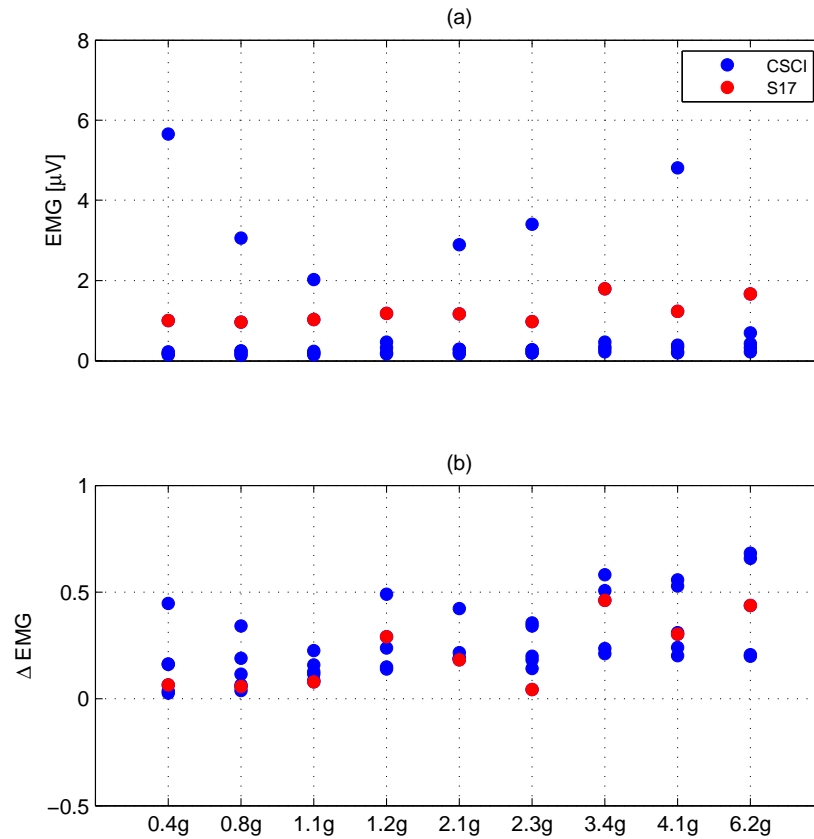


Figure 6.10: TA EMG response of Subject S17 during WBV. The neuromuscular response is presented as absolute EMG (a) and ΔEMG (b). The presented data are for all CSCI participants and test conditions. The presented data are average (mean) values for each participant and grouped according platform acceleration (Equation (4.2)). The specific vibration frequency and vibration amplitude conditions used are identifiable by cross-referencing the acceleration values presented on the x-axis to those described in Table 4.6. TA: Tibialis Anterior; EMG: Absolute EMG activity; ΔEMG : Change in EMG relative to baseline.

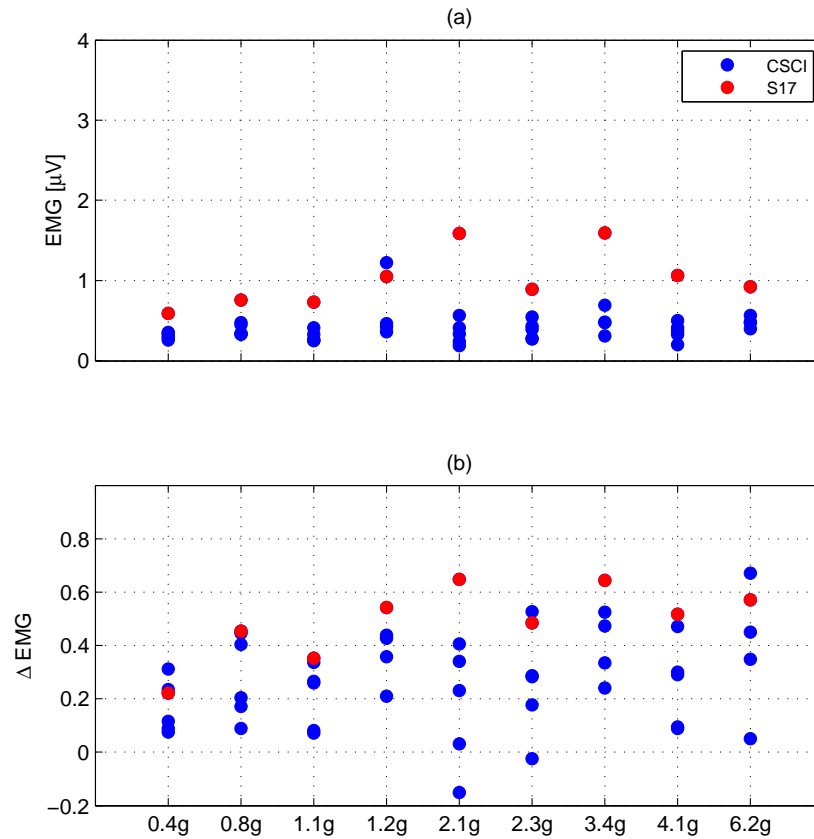


Figure 6.11: M-GA EMG response of Subject S17 during WBV. The neuromuscular response is presented as absolute EMG (a) and Δ EMG (b). The presented data are for all CSCI participants and test conditions. The presented data are average (mean) values for each participant and grouped according platform acceleration (Equation (4.2)). The specific vibration frequency and vibration amplitude conditions used are identifiable by cross-referencing the acceleration values presented on the x-axis to those described in Table 4.6. M-GA: Medial Gastrocnemius; EMG: Absolute EMG activity; Δ EMG: Change in EMG relative to baseline.

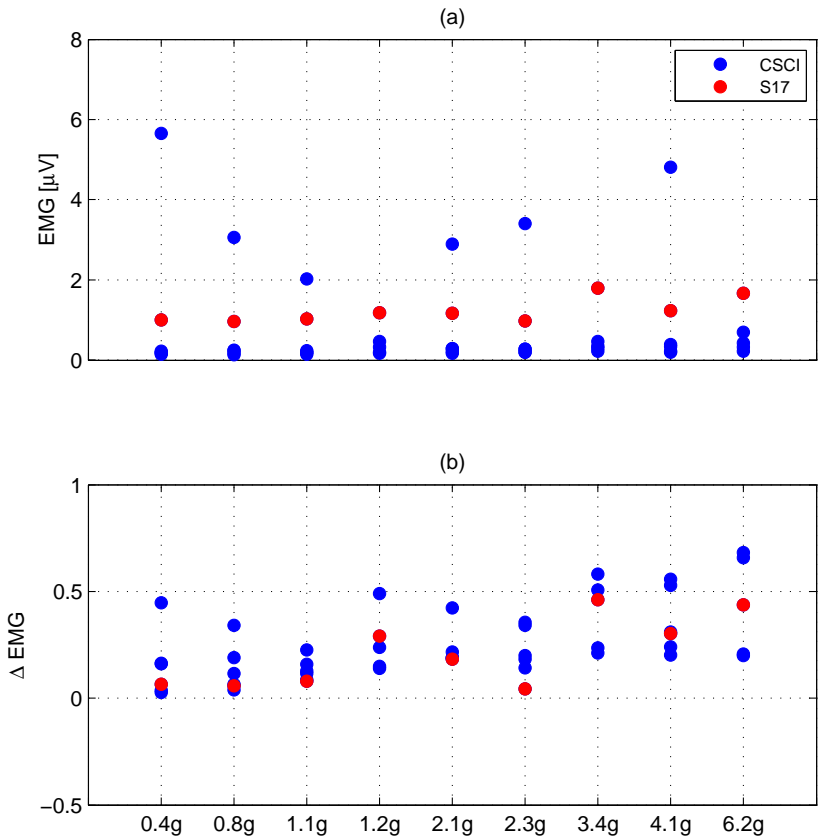


Figure 6.12: VL EMG response of Subject S17 during WBV. The neuromuscular response is presented as absolute EMG (a) and ΔEMG (b). The presented data are for all CSCI participants and test conditions. The presented data are average (mean) values for each participant and grouped according platform acceleration (Equation (4.2)). The specific vibration frequency and vibration amplitude conditions used are identifiable by cross-referencing the acceleration values presented on the x-axis to those described in Table 4.6. VL: Vastus Lateralis; EMG: Absolute EMG activity; ΔEMG : Change in EMG relative to baseline.

There were no notable difference between the group averages of the early-stage SCI participants and Subject S17. Tabulated results of the data presented in the figures above are shown in Table 6.5 & 6.6.

Table 6.5: Comparison of CSCI EMG averages against Subject S17. The tabulated values are the average (mean) EMG values for the CSCI group. CSCI: Motor Complete Spinal Cord Injury; TA: Tibialis Anterior; M-GA: Medial Gastrocnemius; VL: Vastus Lateralis.

Muscle	Group	Platform Acceleration [g]								
		0.4g	0.8g	1.1g	1.2g	2.1g	2.3g	3.4g	4.1g	6.2g
TA	CSCI	1.23	0.80	0.63	0.46	0.83	0.88	0.62	1.19	0.66
	S17	0.99	0.96	1.02	1.17	1.17	0.98	1.79	1.22	1.66
M-GA	CSCI	0.35	0.44	0.37	0.70	0.55	0.46	0.70	0.47	0.56
	S17	0.59	0.75	0.72	1.04	1.58	0.89	1.59	1.05	0.91
VL	CSCI	0.50	0.82	0.55	0.48	0.54	0.66	0.65	0.72	0.46
	S17	0.47	0.47	0.47	0.59	0.44	0.45	0.74	0.47	0.55

Table 6.6: Comparison of CSCI Δ EMG averages against Subject S17. The tabulated values are the average (mean) Δ EMG values for the CSCI group. CSCI: Motor Complete Spinal Cord Injury; TA: Tibialis Anterior; M-GA: Medial Gastrocnemius; VL: Vastus Lateralis; Δ EMG: Change in EMG amplitude relative to baseline.

Muscle	Group	Platform Acceleration [g]								
		0.4g	0.8g	1.1g	1.2g	2.1g	2.3g	3.4g	4.1g	6.2g
TA	CSCI	0.14	0.13	0.13	0.26	0.23	0.21	0.39	0.35	0.43
	S17	0.06	0.05	0.08	0.29	0.18	0.04	0.46	0.30	0.43
M-GA	CSCI	0.17	0.29	0.22	0.39	0.25	0.28	0.44	0.29	0.41
	S17	0.22	0.45	0.35	0.54	0.64	0.48	0.64	0.51	0.57
VL	CSCI	-0.28	-0.11	0.14	0.14	0.07	0.05	0.26	0.22	-0.11
	S17	0.04	0.04	0.03	0.30	0.01	0.00	0.27	0.06	0.13

It was observed that the EMG and Δ EMG values for Subject S17 recorded from the TA and VL were at or near the CSCI group averages. Similarly, the Δ EMG values for Subject S17 recorded from the M-GA were broadly inline with the CSCI group averages also, and only the EMG values of the M-GA were found to be consistently larger than the group averages. Despite being larger however, they were typically of the order of 05 - 1.0 μ V larger, and such a difference is relatively small, particularly when compared to the changes recorded for the ISCI participants. It was thus concluded that the chronicity of injury had little effect upon the magnitude of the acute neuromuscular response of the lower limb to a bout of WBV.

6.1.7 Specific EMG Amplitude Response

The general analysis above confirmed the assumption that the magnitudes of the neuromuscular responses of the ISCI and CSCI participants were significantly different. The differing responses from the different muscle groups shown above were also expected from the analysis carried out previously with the neurologically-intact participants. Vibration was shown to be damped as it was transmitted upwards through the legs and the results from the SCI participants are consistent with that finding. Considering these results in combination with the fact that the CSCI participants were unable to elicit a voluntary muscle contraction during pre-Vibration, whereas the ISCI participants could, it was concluded that the ISCI and CSCI subgroup should be analysed separately hereafter.

The magnitudes of neuromuscular activity during pre-Vibration and Vibration were compared

by plotting average Vibration EMG_{RMS} values against average pre-Vibration EMG_{RMS} values for the different muscle and SCI groups. The results are plotted in Figure 6.13 and a best fit trend line is overlaid the scatter plots, with the slope of the line reported, to highlight the general trends. Note that a slope ≤ 1 indicates that neuromuscular activity was either unchanged or decreased during the Vibration epoch. Table 6.7 summarises the relevant statistics for the trend lines.

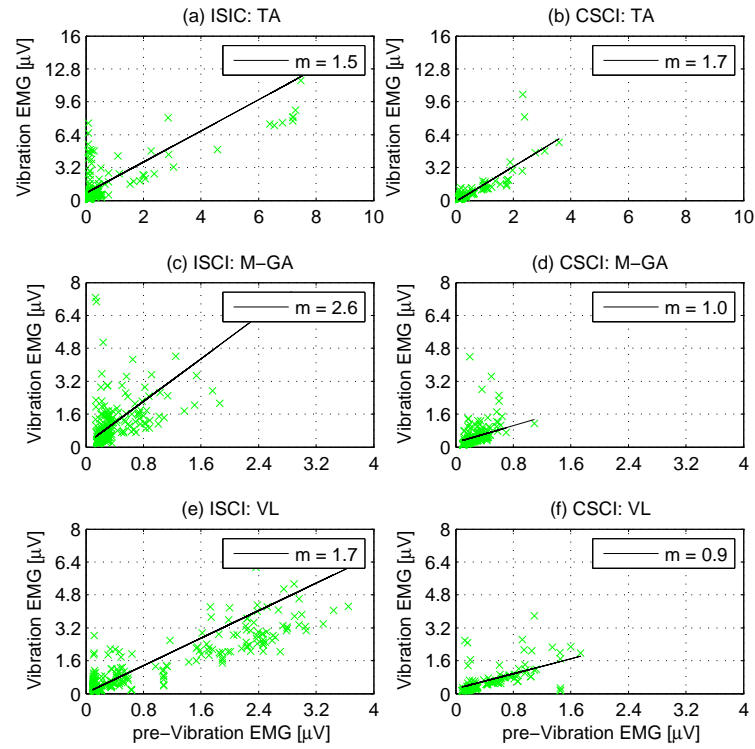


Figure 6.13: Scatter plot of pre-Vibration and Vibration muscle activity with best-fit trend line. Best-fit line assessed using Pearson's correlation coefficient. TA: Tibialis Anterior, M-GA: Medial Gastrocnemius, and VL: Vastus Lateralis, CSCI: motor-complete SCI, ISCI: motor-incomplete SCI, SCI: spinal cord injury.

Table 6.7: Trend-line statistics describing relationship between pre-Vibration and Vibration muscle activity. Best-fit line assessed using Pearson’s correlation coefficient. R-value: Pearson’s best fit correlation; P-value: statistical significance.

SCI Group	Muscle	Slope	Intercept	R-value	P-value
ISCI	TA	1.5	0.7	0.6	<0.01
	M-GA	2.6	0.2	0.8	<0.01
	VL	1.7	0.0	0.7	<0.01
CSCI	TA	1.7	-0.1	0.7	<0.01
	M-GA	1.0	0.2	0.2	<0.01
	VL	0.9	0.2	0.3	<0.01

The slope of the trend line indicates the change in muscle activity expected to occur with vibration. In all cases there was a positive and statistically-significant increase in muscle activity for the ISCI participants. In contrast, this trend was only seen in the TA of the CSCI participants. There were some instances where M-GA and VL activity increased but in general these increases were small. It was therefore concluded that the type of response was dependent on the muscle group and classification of SCI. This confirmed that the initial conditions were different for the muscle groups (e.g. pre-contraction in the muscle) and affirmed that the SCI groups should be assessed separately.

The amplitude responses of each individual muscle group to WBV are presented in the following sub-sections. The amplitude of the muscle responses were quantified in two ways: (i) by describing the overall magnitude of neuromuscular activity by computing the RMS and calculating the average for the Vibration epoch and (ii) by describing the relative change in neuromuscular activity to a baseline condition (Equation (4.12)). The data are presented for all EMG which were not rejected as outliers. The data were grouped according to classification of injury and platform acceleration (Equation (4.13)). The 0.0g (pre-Vibration) condition is representative of baseline. The specific vibration frequency and vibration amplitude conditions used may be determined by referencing the acceleration values described on the x-axis to those defined in Table 4.6.

Supporting statistical analyses are provided in associated tables throughout. A multi-way ANOVA of all EMG_{RMS} and ΔEMG data was carried out for both SCI groups. The independent grouping factors were vibration frequency and vibration amplitude.

Tibialis Anterior

WBV resulted in an increase in TA neuromuscular activity. ΔEMG generally increased with platform acceleration, and the increases were larger for the ISCI participants. ΔEMG was typically between 35 and 87% for the ISCI participants, which equated to an EMG increase between 0.32 and $0.70\mu\text{V}$. By comparison, ΔEMG was typically between 6 and 39% for the CSCI participants, which equated to an EMG increase between 0.20 and $0.43\mu\text{V}$. The statistical significance of these findings are outlined in Table 6.8.

In summary, the relative change in activity at the 3mm condition was significantly larger than the change at the 1mm and 2mm conditions for both the ISCI and CSCI participants. In addition, the relative change in activity during 27Hz vibration was significantly larger than the relative change at the 12Hz condition for both the ISCI and CSCI participants.

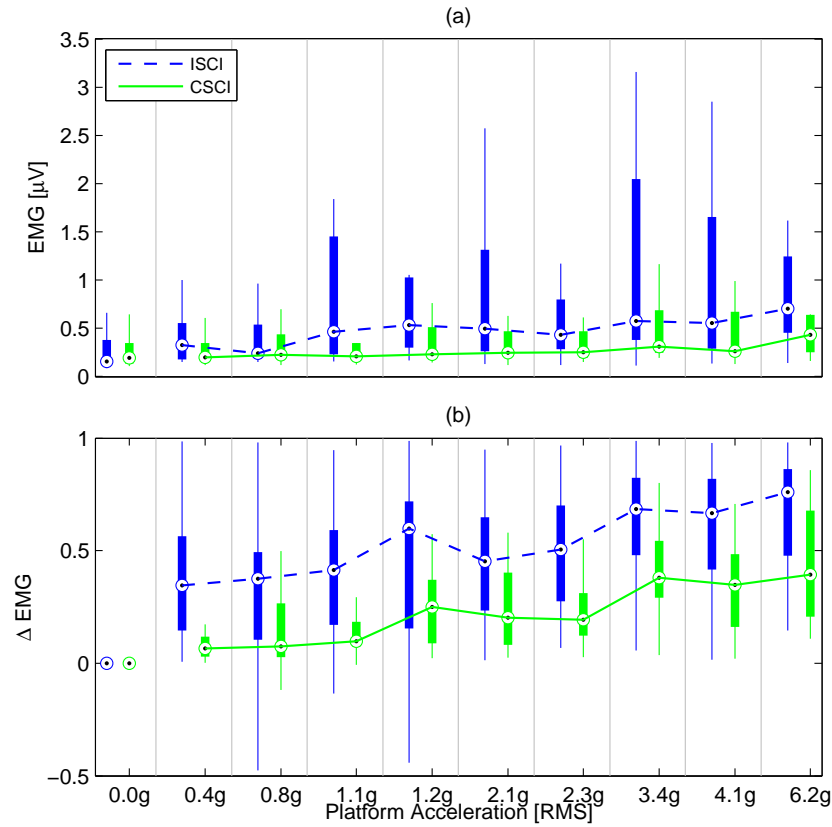


Figure 6.14: Amplitude response of Tibialis Anterior to WBV. The neuromuscular response is presented as absolute EMG (a) and ΔEMG (b). The presented data are for all participants and test conditions. The data were grouped according the platform acceleration (Equation (4.2)) and classification of SCI. Pre-Vibration data are presented for reference purposes and can be identified as the 0.0g condition. The specific vibration frequency and vibration amplitude conditions used are identifiable by cross-referencing the acceleration values presented on the x-axis to those described in Table 4.6.

Table 6.8: Statistics table of TA results. A multi-way ANOVA was used to identify significant changes in EMG and Δ EMG. The independent factors used were vibration frequency and vibration amplitude. The interaction between these terms was also investigated. Where statistical significance was identified, a post-hoc multiple comparisons procedure with Bonferroni correction was used to identify significantly different pairs. EMG: EMG recorded during WBV; Δ EMG: Change in activity relative to baseline (Equation (4.12)); TA: Tibialis anterior; F: ANOVA F-statistic; ANOVA: Analysis of Variance; DF: Degrees of freedom; p: P-value; Gu_{Gl} : Pairs identified to be significantly different in post-hoc analysis; Gu: Upper group; Gl: Lower group.

SCI Group	Parameter	Δ EMG			EMG _N		
		F (DF)	p	Gu_{Gl}	F (DF)	p	Gu_{Gl}
ISCI	Frequency	5.91 (2)	≤ 0.01	27 ₁₂	0.87 (2)	=0.42	-
	Displacement	7.52 (2)	≤ 0.01	3 ₁ ; 3 ₂	1.73 (2)	=0.18	-
	Interaction	0.69 (4)	=0.60	-	0.11 (4)	=0.98	-
CSCI	Frequency	12.70 (2)	≤ 0.01	27 ₂₀ ; 27 ₁₂	0.20 (2)	=0.82	-
	Displacement	19.46 (2)	≤ 0.01	3 ₁ ; 3 ₂	0.16 (2)	=0.85	-
	Interaction	0.94 (4)	=0.44	-	0.32 (4)	=0.87	-

Table 6.9 compares these results with the relative changes in neuromuscular activity of the the neurologically-intact participants to baseline. In every case, Δ EMG was larger for the ISCI (50% pBWS) and CSCI (100% pBWS) participants when compared to the neurologically intact participants under similar experimental conditions.

Table 6.9: Comparison of the average change in TA neuromuscular activity for the neurologically-intact (AB) and SCI participants for each platform acceleration and level of pBWS. The tabulated values are the average (mean) changes in neuromuscular activity relative to baseline (pre-Vibration epoch) and all tabulated values are presented as percentages (Δ EMG $\times 100$). pBWS: partial Body Weight Support; ISCI: Motor Incomplete Spinal Cord Injury; CSCI: Motor Complete Spinal Cord Injury; TA: Tibialis Anterior.

Group	pBWS	Platform Acceleration [g]								
		0.4g	0.8g	1.1g	1.2g	2.1g	2.3g	3.4g	4.1g	6.2g
AB	0	24.06	24.06	25.91	40.31	41.94	36.89	46.96	48.08	60.96
AB	50	9.49	17.35	12.87	30.20	26.25	21.51	30.17	26.93	36.06
ISCI	50	37.60	34.42	40.61	48.61	45.14	45.36	63.03	58.76	66.30
AB	100	9.73	9.15	9.06	11.35	14.61	14.24	15.15	14.34	25.57
CSCI	100	12.28	12.99	12.99	26.12	23.29	21.19	39.90	34.14	43.69

Medial Gastrocnemius

Similar increases in ΔEMG were found for the M-GA. The increases were larger for the ISCI participants and ΔEMG was between 32 and 68%, this equated to a 0.39 to $1.39\mu\text{V}$ increase in EMG. In comparison, ΔEMG was typically between 12 and 51% for the CSCI participants, which equated to an EMG increase between 0.36 and $0.42\mu\text{V}$. These increases in neuromuscular activity were statistically significant at the 3mm condition for both the ISCI and CSCI participants when compared to the 1mm condition. The relative increase at the 27Hz condition was significantly larger than that for the 12Hz condition for the ISCI participants. The results of the statistical analysis are summarised in Table 6.10.

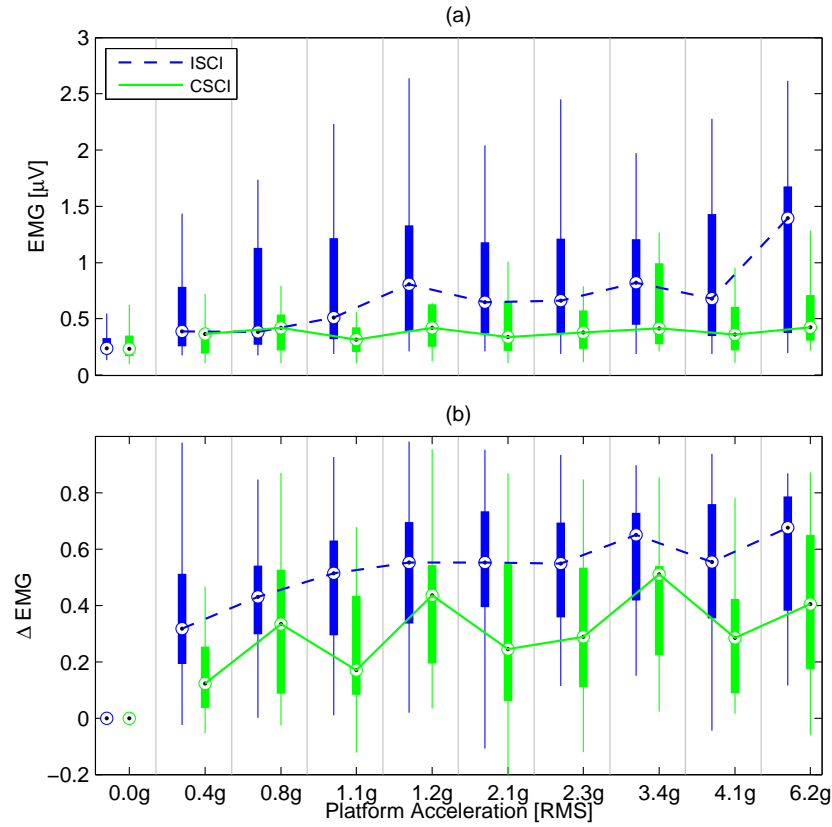


Figure 6.15: Amplitude response of Medial Gastrocnemius to WBV. The neuromuscular response is presented as absolute EMG (a) and ΔEMG (b). The presented data are for all participants and test conditions. The data were grouped according the platform acceleration (Equation (4.2)) and classification of SCI. Pre-Vibration data are presented for reference purposes and can be identified as the 0.0g condition. The specific vibration frequency and vibration amplitude conditions used are identifiable by cross-referencing the acceleration values presented on the x-axis to those described in Table 4.6.

Table 6.10: Statistics table of GA results. A multi-way ANOVA was used to identify significant changes in EMG and Δ EMG. The independent factors used were vibration frequency and vibration amplitude. The interaction between these terms was also investigated. Where statistical significance was identified, a post-hoc multiple comparisons procedure with Bonferroni correction was used to identify significantly different pairs. EMG: EMG recorded during WBV; Δ EMG: Change in activity relative to baseline (Equation (4.12)); M-GA: Medial gastrocnemius; F: ANOVA F-statistic; ANOVA: Analysis of Variance; DF: Degrees of freedom; p: P-value; Gu_{GI} : Pairs identified to be significantly different in post-hoc analysis; Gu: Upper group; Gl: Lower group.

SCI Group	Parameter	Δ EMG			EMG _N		
		F (DF)	p	Gu_{GI}	F (DF)	p	Gu_{GI}
ISCI	Frequency	4.58 (2)	≤ 0.05	27 ₁₂	0.61 (2)	=0.54	-
	Displacement	3.09 (2)	≤ 0.05	3 ₁	1.70 (2)	=0.19	-
	Interaction	0.42 (4)	=0.80	-	0.29 (4)	=0.89	-
CSCI	Frequency	0.51 (2)	=0.60	-	0.09 (2)	=0.91	-
	Displacement	8.09 (2)	≤ 0.01	3 ₁	2.97 (2)	=0.05	-
	Interaction	0.30 (4)	=0.88	-	0.76 (4)	=0.56	-

Table 6.11 compares these results with the relative changes in neuromuscular activity of the the neurologically-intact participants to baseline. At the lower intensity vibration levels, typically at platform accelerations of 2.3g or less, the Δ EMG values of the SCI participants were larger than the responses of the neurologically intact participants under similar experimental conditions. However this trend reversed as platform acceleration increased, particularly at platform accelerations greater than 4g. It was hypothesised that the neurologically intact participants were able to successfully maintain their posture and position on the vibration platform due to their greater capacity for motor control, therefore resulting in the high intensity vibration being effectively imparted on them. By contrast, the decreased functional capacity of the SCI participants, and particularly that of the CSCI participants, possibly increased the likelihood of their feet moving during the Vibration epoch and therefore reducing the transmission of vibration.

Table 6.11: Comparison of the average change in M-GA neuromuscular activity for the neurologically-intact (AB) and SCI participants for each platform acceleration and level of pBWS. The tabulated values are the average (mean) changes in neuromuscular activity relative to baseline (pre-Vibration epoch) and all tabulated values are presented as percentages (Δ EMG $\times 100$). pBWS: partial Body Weight Support; ISCI: Motor Incomplete Spinal Cord Injury; CSCI: Motor Complete Spinal Cord Injury; M-GA: Medial Gastrocnemius.

Group	pBWS	Platform Acceleration [g]								
		0.4g	0.8g	1.1g	1.2g	2.1g	2.3g	3.4g	4.1g	6.2g
AB	0	15.70	32.53	40.45	38.56	55.89	48.11	58.09	70.80	71.44
AB	50	26.70	45.01	26.83	49.45	44.85	44.10	53.08	53.72	70.51
ISCI	50	35.72	40.60	47.13	52.07	52.34	52.21	54.63	52.86	58.30
AB	100	17.16	20.71	21.63	27.30	31.35	23.61	37.27	41.94	55.65
CSCI	100	18.36	31.37	24.15	39.52	28.75	31.77	44.37	31.16	41.78

Vastus Lateralis

In general, the EMG response of the VL to WBV was similar to that of the M-GA even though the relative change in activity was smaller. ΔEMG was between 7 and 87% for the ISCI participants, and this equated to a 0.43 to 1.23 μV increase in EMG amplitude. In comparison, ΔEMG was typically between 5 and 27% for the CSCI participants, which equated to an EMG amplitude of 0.28 to 0.52 μV . There was no instance where the increase in neuromuscular activity for any of the participants was significant and these results are summarised in Table 6.12.

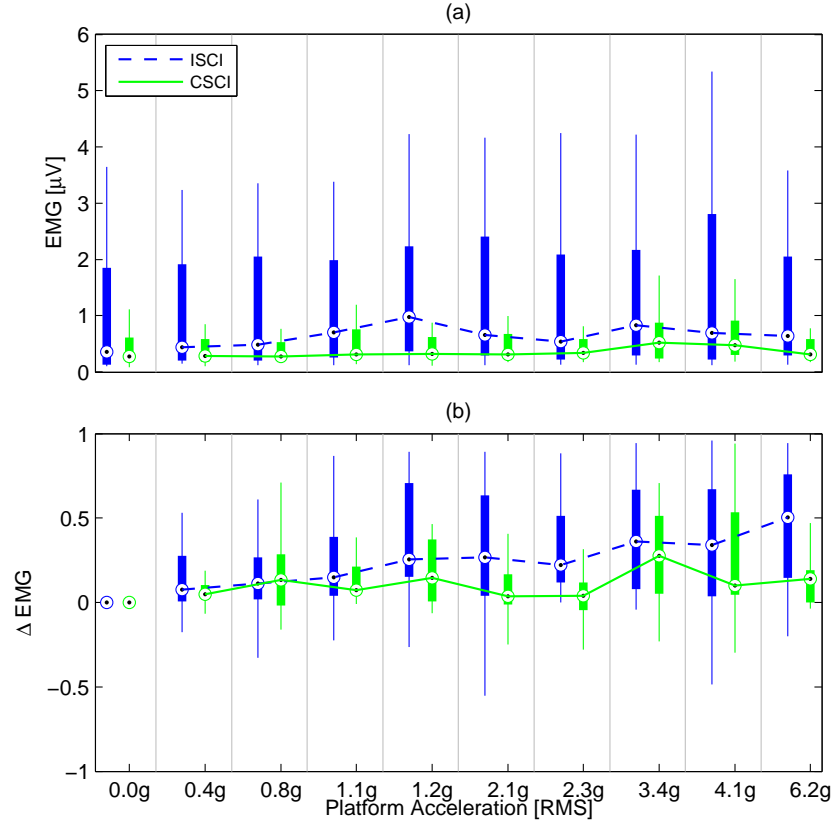


Figure 6.16: Amplitude response of Vastus Lateralis to WBV. The neuromuscular response is presented as absolute EMG (a) and ΔEMG (b). The presented data are for all participants and test conditions. The data were grouped according the platform acceleration (Equation (4.2)) and classification of SCI. Pre-Vibration data are presented for reference purposes and can be identified as the 0.0g condition. The specific vibration frequency and vibration amplitude conditions used are identifiable by cross-referencing the acceleration values presented on the x-axis to those described in Table 4.6.

Table 6.12: Statistics table of VL results. A multi-way ANOVA was used to identify significant changes in EMG and Δ EMG. The independent factors used were vibration frequency and vibration amplitude. The interaction between these terms was also investigated. Where statistical significance was identified, a post-hoc multiple comparisons procedure with Bonferroni correction was used to identify significantly different pairs. EMG: EMG recorded during WBV; Δ EMG: Change in activity relative to baseline (Equation (4.12)); VL: Vastus lateralis; F: ANOVA F-statistic; ANOVA: Analysis of Variance; DF: Degrees of freedom; p: P-value; Gu_{GI} : Pairs identified to be significantly different in post-hoc analysis; Gu: Upper group; Gl: Lower group.

SCI Group	Parameter	Δ EMG			EMG _N		
		F (DF)	p	Gu_{GI}	F (DF)	p	Gu_{GI}
ISCI	Frequency	1.36 (2)	=0.26	-	1.18 (2)	=0.31	-
	Displacement	2.60 (2)	=0.08	-	1.07 (2)	=0.34	-
	Interaction	0.67 (4)	=0.61	-	0.18 (4)	=0.95	-
CSCI	Frequency	1.17 (2)	=0.31	-	0.30 (2)	=0.74	-
	Displacement	0.23 (2)	=0.79	-	1.13 (2)	=0.32	-
	Interaction	1.06 (4)	=0.38	-	0.36 (4)	=0.84	-

Table 6.13 compares these results with the relative change in neuromuscular activity of the neurologically-intact participants to baseline. The Δ EMG values from the ISCI participants were generally larger or comparable to the values from the neurologically intact participants for all platform acceleration levels. By contrast there were a large number of instances where the response of the CSCI participants actually decreased during the Vibration epoch. This is most likely due to the sensitivity of the Δ EMG calculation for the CSCI participants. The level of activity during the pre-Vibration epoch was always small by comparison to the ISCI and neurologically intact participants. Any small change in neuromuscular output would appear magnified when viewing the Δ EMG value and comparing to the other participant groups. It is therefore necessary to view the absolute EMG magnitudes also (Figure 6.16) where it can be seen that CSCI activity during the pre-Vibration and Vibration epochs was always small.

Table 6.13: Comparison of the average change in VL neuromuscular activity for the neurologically-intact (AB) and SCI participants for each platform acceleration and level of pBWS. The tabulated values are the average (mean) changes in neuromuscular activity relative to baseline (pre-Vibration epoch) and all tabulated values are presented as percentages (Δ EMG \times 100). pBWS: partial Body Weight Support; ISCI: Motor Incomplete Spinal Cord Injury; CSCI: Motor Complete Spinal Cord Injury; VL: Medial Gastrocnemius.

Group	pBWS	Platform Acceleration [g]								
		0.4g	0.8g	1.1g	1.2g	2.1g	2.3g	3.4g	4.1g	6.2g
AB	0	14.18	17.57	18.11	19.42	25.20	21.78	25.33	28.32	31.95
AB	50	17.01	24.77	18.93	22.44	8.11	21.60	28.41	22.75	27.51
ISCI	50	2.60	7.52	24.30	37.48	29.85	22.35	31.53	21.99	35.66
AB	100	17.95	26.84	19.18	25.84	29.85	23.12	23.19	23.77	28.36
CSCI	100	-37.87	-20.41	14.99	17.37	10.00	4.35	27.02	26.46	-17.16

In summary, analysis of the individual muscles generally reflected those trends identified in the general analysis (Section 6.1.5). Vibration frequency and vibration amplitude each

elicited an increase in neuromuscular activity, but the magnitude of the increase was not consistent across each of the muscle groups. Overall, both ΔEMG and EMG were larger across all test conditions for the ISCI participants and in general the neuromuscular response increased with platform acceleration.

The VL and M-GA were the most active muscle groups, each exhibiting similar values of ΔEMG and EMG, and the TA was the least active. ΔEMG is the parameter of most interest from this analysis. It was not possible to perform intermuscular comparisons of the EMG amplitude results from the SCI participants, because it was not possible to normalise the EMG to a MVC. Normalisation to MVC was not done because: (i) it was not possible to perform an MVC with the CSCI participants and (ii) those MVC recorded from the ISCI participants were highly variable and therefore an unreliable indication of the maximum capacity of the muscle. The largest changes in activity were found in those muscles which were in closest proximity to the vibration platform, and in the event that there was a change in EMG activity there was a corresponding change in ΔEMG also.

6.1.8 EMG Vibration Cycle Analysis

This analysis has focussed on the steady-state neuromuscular response to WBV up to this point. An ideal, steady-state EMG would therefore be ‘flat’ during the entire period of vibration, therefore implying that the neuromuscular response over a period of 6s for example would be equivalent to the response over a single cycle of vibration. However, a review of time series EMG data showed that this was not the case. In practice, fluctuations in the EMG were compensated for by averaging the EMG over a set period by calculating the RMS. Some characteristics of the EMG were lost by doing this, such as those identified previously by Ritzmann *et al.* [88]. The evidence suggested was the occurrence of a peak in the neurological response which was akin to a short latency reflex and a similar phenomenon was identified in this study, see Section 5.1.6, for the neurologically intact participants. A Vibration Cycle Analysis was carried out to determine if the same trends occurred in an SCI population.

General Analysis

A vibration cycle analysis was carried out on every EMG that met the inclusion criteria described in Section 4.2.8 and the results are summarised in Figure 6.17. The vibration cycle data from each test were averaged for all participants, tests, and test epoch: pre-Vibration, Vibration, post-Vibration. The averaged data are then grouped according to: vibration frequency, vibration amplitude, muscle group and classification of SCI, and then normalised to the maximum response. This was done to determine qualitatively which of these factors was most likely to influence a temporal relationship between neuromuscular activity and a single cycle of vibration.

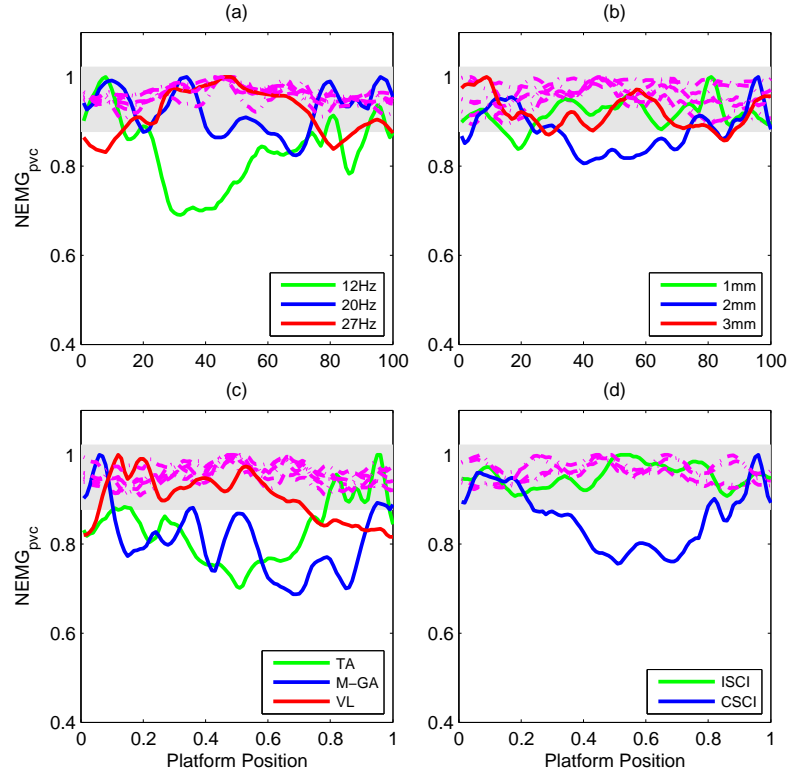


Figure 6.17: Summary of vibration cycle analysis for all EMG. The results were grouped for all participants, tests and test epoch. Pre-Vibration and post-Vibration data are indicated by dashed lines and are enclosed within the light grey banded area. Vibration data are indicated by solid lines. The grouping factors were: vibration frequency (a), vibration amplitude (b), muscle group (c), and classification of SCI (d). Grouped and averaged data are normalised maximum. $NEMG_{pvc}$: Per vibration cycle of the EMG normalised to its maximum.

No significant fluctuation or change in neuromuscular activity was observed for those EMG recorded during the pre-Vibration and post-Vibration epoch as expected, and were therefore excluded from further analysis. Some fluctuation of the EMG grouped according to vibration amplitude and classification of injury were shown but the changes were erratic, relatively small by comparison, and difficult to distinguish when compared to those EMG that were grouped according to the muscle group and vibration frequency. The data grouped according to muscle group and vibration frequency were erratic and difficult to distinguish also but were analysed further on the basis that the magnitude of these signals were larger than those data grouped according to vibration amplitude and injury classification.

Specific Analysis

The analysis was repeated using those EMG which were repeatable between tests and were recorded during the Vibration epoch only. Pearson's correlation coefficient was used to determine if the EMG from each of the testing sessions were sufficiently 'similar'. Both output EMG were rejected if: (i) the correlation between the two EMG was non-significant ($p \geq 0.05$) or (ii) very poor or negative ($r \leq 0.1$). All retained data were averaged for all participants and grouped according to muscle group, leg, classification of SCI and vibration frequency. The

results are shown for the CSCI participants only in Figure 6.18 because none of the EMG recorded from the ISCI participants met the inclusion criteria outline above.

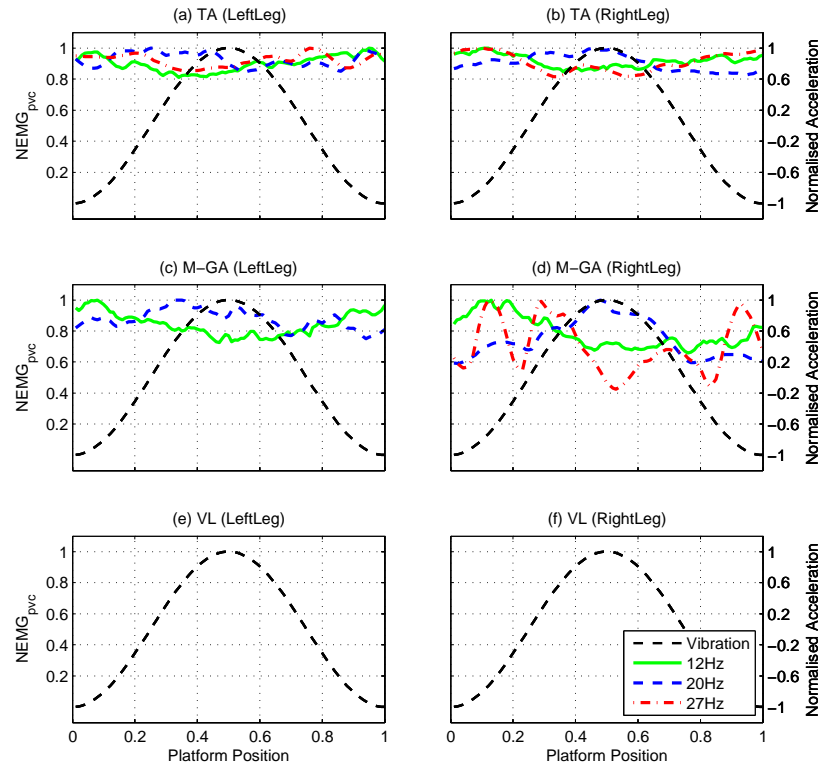


Figure 6.18: Results of vibration cycle analysis for EMG from the CSCI participants recorded during WBV only. The results were averaged for all CSCI participants and tests, and grouped according to vibration frequency and muscle group. Grouped and averaged data were normalised to the respective maximum values. The top panels show the results for the TA recorded from the left (a) and right (b) legs, the middle panels shows the results for the M-GA from the left (c) and right (d) legs, and the bottom panels show the results for the VL from the left (e) and right (f) legs. Note that no data is shown for the VL because none of the EMG met the inclusion criteria outlined for this test. $NEMG_{pvc}$: Per vibration cycle of the EMG normalised to its maximum; TA: Tibialis Anterior; M-GA: Medial Gastrocnemius; VL: Vastus Lateralis.

No temporal relationship between platform position and neuromuscular response was identified for the ISCI participants. However, while some relationships were identified for the CSCI participants, the changes were small in magnitude.

6.2 Discussion

Evidence supporting the application of WBV in SCI rehabilitation is limited, and it has been applied with limited success using a tilt-table and from a seated position [87, 115]. The results from this study were the first to demonstrate the application of WBV to both ISCI and CSCI patients in a standing position, with the support of a pBWS system. The feasibility of this system was confirmed, as positive changes in neuromuscular output were found and directly attributed to the application of vibration. A range of low to high frequencies and amplitudes were systematically assessed and directly compared with normative data recorded from a neurologically intact population. Furthermore, the normative data gathered from the neurologically intact participants was the first to systematically quantify the effect of reduced self-supported body weight on the neuromuscular response during WBV - rather than increasing load as is typically done. The findings from the tests at 0% pBWS were consistent with those reported in the literature, and the findings at 50% and 100% pBWS have added to the understanding of how leg muscles respond to vibration at reduced body loads. A novel analysis approach then allowed for a detailed investigation of both neurologically intact and SCI EMG over a single cycle of vibration. New evidence here suggests that peak platform acceleration is central to eliciting a peak neuromuscular response.

A systematic approach was carried out to quantify the presence of vibration artefact in EMG recorded during WBV with statistical analysis supporting the number of harmonic frequencies which should be removed. The results from this study suggest that a higher proportion of artefact was present in the EMG than that reported by others in the literature [107, 126], however methodological differences between the studies exist and were highlighted in greater detail in the literature review (Section 3.4). Some have removed artefact simply due to the presence of atypical EMG frequency spectra [72, 107–110, 114, 115, 125], meanwhile others have contested that vibration artefact is present at all [88]. Despite reports to the contrary, firm supporting evidence, in the form of a case study, was presented in this thesis which identified the presence of vibration artefact and highlighted the requirement for its removal.

The overall objectives of this study were the same as those for the general population. To recap, the technical objectives were: (i) determine the feasibility of WBV-pBWS in a SCI population, (ii) to quantify the transmission of vibration from the WBV platform to the lower limbs, and (iii) to estimate the proportion of vibration artefact in each EMG. The physiological objectives were: (i) to measure and quantify the magnitude of the acute, steady-state neuromuscular response of the lower limbs to WBV-pBWS and to compare the response of the SCI population with the response of a general population with no neurological deficit and (ii) to investigate the temporal relationship between neuromuscular activity and the position of the WBV platform. These technical and physiological objectives are discussed separately in the following sections.

6.2.1 Physiological

The specific physiological objectives outlined at the beginning of this chapter are addressed below. These objectives were achieved by analysing the acquired EMG data in a similar

manner to that done for the neurologically intact population and therefore allowed for a direct comparison of the results from the different populations. Recall, that the neurologically intact population were tested with all levels of pBWS whereas the SCI participants were tested with a level of support appropriate to their functional capability. In this study all of the ISCI participants were tested with 50% pBWS and the CSCI participants were tested with 100% pBWS.

Δ EMG was comparable across all sampled populations because this outcome measure was not influenced by the level of functional capability of the participant, though it was sensitive to small levels of baseline activity, which was the case for the CSCI participants. For a given set of experimental conditions, the magnitude of change in neuromuscular activity of the ISCI participants from pre-Vibration to Vibration was equivalent or in excess of that achieved by the neurologically intact participants. Specific values are discussed in the following section, but typical changes for the neurologically intact participants were between 6.9 - 29.7% (TA), 22.6 - 73.7% (M-GA) and 15.5 - 24.4% (VL). Equivalent results for the ISCI participants were between 34.7 - 76% (TA), 31.8 - 67.7% (M-GA) and 7.5 - 50.3% (VL).

These relative changes were normalised to MVC for the neurologically intact participants and were found to be between 0.7 - 1.2% MVC (TA), 3.6 - 12.6% MVC (M-GA) and 9.4 - 10.9% MVC (VL), and were considered to be relatively small when compared to the changes at 0% pBWS. At 0% pBWS, the overall changes in activity were between 1.1 - 2.0 MVC (TA), 5.9 - 18.9 MVC (M-GA) and 17.7 - 26.5 MVC (VL). The findings in this study and those reported in the literature are in good agreement. The same relationship between the magnitude of the neuromuscular response with vibration frequency and vibration amplitude was found, though the overall magnitudes of contraction reported here were smaller [72, 76, 90, 109]. This is likely a consequence of the robust off-line EMG classification procedure used here to reject outlying data and artefact, in addition to methodological differences which included supported load, applied vibration frequency/amplitudes and posture. A similar investigation carried out by Pollock *et al.* [72] reported equivalent values between 2 - 32% (approx.) MVC (TA), 5 - 42% MVC (approx.) and (M-GA) and 5 - 18% MVC (approx.) (RF - VL was not reported on). In general, the smaller EMG amplitudes reported in this study are most likely accounted for by differing testing methodologies, for example Pollock *et al.* [72] applied vibration amplitudes at 2.5 & 5.5mm in a straight leg position, compared to 1, 2, & 3mm in a flexed knee position in this study. Roelants *et al.* [90] on the other hand increased the magnitude of neuromuscular response during vibration by increasing pre-contraction by prescribing unilateral static squats, while Hazell *et al.* [109] simply increased the supported load. Others have suggested that the maximal response of the VL occurs at 30Hz [76], however these findings could not be corroborated using this data set because the WBV platform used here was limited to a maximum vibration frequency of 27Hz.

Thus, despite the overall changes being considered as relatively small for the neurologically intact participants, they would be considered large for a SCI participant. Furthermore, the magnitudes of the neuromuscular responses found for those with a SCI were larger than those found elsewhere. Neither Herrero *et al.* [115] or Chang *et al.* [87] identified an increased neuromuscular response from an SCI population. Herrero *et al.* [115] applied vibration on

a tilt-table, rather than in an upright and standing position, and was thus unlikely to have applied an adequate ground reaction force to ensure a constant and reliable contact with the vibration platform. Chang *et al.* [87] on the other hand applied vibration from a seated position and concluded that the absence of an increased neuromuscular response was due to a lack of vestibulospinal activity, suggesting that a postural control mechanism mediates the acute neuromuscular response. However, data gathered as part of this work did not support this conclusion, and evidence from this study suggested that platform acceleration mediated the peak neuromuscular response. This observation was in line with findings from others [88].

It was thus determined that, given the similarities between ΔEMG for both the SCI and neurologically intact participants, it is therefore plausible that similar increments in actual muscle contraction would occur for the SCI participants also. The conclusions of a comparison between ΔEMG values from the neurologically intact participants with 100% pBWS and CSCI participants are the same. Values in the ranges between 4 - 40% (TA), 5 - 68% (M-GA) and 5 - 28% (VL) were found for both populations. These values compare favorably with those reported above but they were typically associated with a muscular contraction $<8\%$ MVC in the M-GA, and $<2\%$ MVC in the TA and VL for the neurologically intact participants. Furthermore, a review of the raw EMG data from the CSCI participants demonstrated the sensitivity of the ΔEMG calculation to small baseline levels of neuromuscular activity, as in many cases the EMG amplitude either remained the same during the Vibration epoch, or decreased to a smaller level than that recorded for the pre-Vibration epoch. It is possible that the decrease in neuromuscular activity was the result of some neural inhibition, however it is more likely a consequence of the offline analysis. Filtering of the data to remove artefact would have a comparatively large effect on CSCI EMG signals which would be relatively small to begin with.

In any case, the same rationale that applied to the ISCI participants above, applies again for the CSCI participants here. The CSCI participants in particular have no capacity for any volitional contraction of muscle, therefore any induced contraction would have a potential therapeutic benefit, however small the contraction may be.

The trends in the change in neuromuscular activity with vibration frequency and amplitude, both of which are components of acceleration, are in agreement with those observed elsewhere, where there was an increase in neuromuscular output with both vibration frequency and amplitude [72, 73, 76, 109]. However, the analysis carried out for this thesis went further and analysed the neuromuscular response over a single cycle of vibration. The novel analysis methodology averaged the EMG to allow the identification of the most likely pattern of neuromuscular activity over the course of a single vibration cycle. Maxima were observed in the EMG of the TA, M-GA and VL of the neurologically intact participants and the latency of the maxima were traced back to peak platform acceleration in the preceding vibration cycle.

Such maxima were identified previously by Ritzmann *et al.* [88] who concluded that these maxima were short latency reflexes being elicited by some component of motion of the vibration platform. The results of the analysis carried out in this study neither confirm nor refute this conclusion though the latencies identified in this study were longer than those

reported in the literature, noting that in this study the TA, M-GA and VL were assessed whereas Ritzmann *et al.* assessed the TA, RF and soleus. These results therefore imply that the maxima were induced by reflex activity, probably by peak platform acceleration, but were not short latency reflexes. Such maxima were not successfully identified for the SCI participants. Ritzmann *et al.* showed that the stretch receptor was central to the elicited neuromuscular response to WBV (Section 3.2). It is possible that the failure to identify the maxima in the SCI EMG was due to the cord injury itself, whereby the elicited afferents from the stretch receptor were either being misinterpreted, were of insufficient intensity to depolarise the α -motoneuron, or the neuromuscular response was simply too small to be detected reliably. Furthermore, it is known that SCI severely disrupts normal spinal reflexes and pathways, e.g. stretch receptor activation resulting in clonic spasm.

The physiological objectives specific to this chapter are discussed in greater detail below.

EMG Amplitude Response

The analysis techniques used to quantify the amplitudes of the EMG recorded from the neurologically intact participants were repeated for the SCI participants³. Analysis confirmed that WBV resulted in a significant increase in the amplitude of the EMG. Having established that WBV caused an increase in neuromuscular activity, the magnitude of this increase was assessed. The amplitude was quantified in two ways: Δ EMG (relative change in EMG from baseline) and *EMG* (simply the maximum amplitude of the *EMG_{RMS}*).

Δ EMG and *EMG* were pooled for the Vibration epoch only and grouped according to the four main factors under investigation in this study: vibration frequency, vibration amplitude, muscle group and classification of SCI. The differences in the magnitudes of the responses were significantly different for each of these grouping factors. The change in neuromuscular activity was significantly larger for the ISCI participants during the Vibration epoch than it was for the CSCI participants. Δ EMG was typically 48% for the ISCI participants and 15% for the CSCI participants. This supported the hypothesis that some degree of muscle activity or contraction prior to vibration significantly affected the magnitude of the response during vibration.

Vibration frequency and amplitude each resulted in a significant increase in neuromuscular activity, where typical positive Δ EMG changes were between 21 - 43%. These increases were equivalent to an *EMG* change of 0.37 - 0.54 μ V. At the outset, these overall increases in activity were small, however it is worth noting that these average values include the responses from both SCI groups, and it is known that the response of the CSCI group were significantly smaller than the ISCI group.

The largest changes occurred in the shank muscles, where typical Δ EMG were 33% and 45% in the TA and GA respectively during WBV. The corresponding changes in the VL were considerably smaller at 16%. However as with the neurologically intact population, though

³Recall that the principal method for normalising EMG was to calculate Δ EMG. This was particularly relevant for the SCI participants because it was not possible to record a reliable MVC due to a combination of factors which included classification and severity of injury, and time since injury.

the relative change in activity was smaller, the absolute level of activity of the VL appeared to be larger.

This was supported by comparing the level of neuromuscular activity during the pre-Vibration and Vibration epochs. Figure 6.13 shows this relationship for each muscle and SCI group respectively. In all cases there was a positive (i.e. slope >1) and statistically significant change in muscle activity for the ISCI participants. However, these same increases were only seen in the TA for the CSCI participants. These data confirm that there was an increased response to WBV, but that the increase was predominantly found for the ISCI participants. In addition, it was also noted that the type of response for each muscle group was different, as indicated by the slope of the trend line.

EMG and ΔEMG were assessed for each individual muscle and SCI group and plotted in Figures 6.14 - 6.16. The same general trends were observed for all muscle groups and the salient results from these figures are summarised in Table 6.14 below for reference. These results are supported by those presented in Figure 6.13 where it was shown that there was a steeper slope for the shank muscles when compared to those increases for the thigh muscles, and that the ISCI participants generally exhibited a larger response.

Table 6.14: Range of typical *EMG* and ΔEMG values for each muscle and SCI group. *EMG* and ΔEMG data were grouped according to classification of SCI and platform acceleration for all participants and tests. The largest and smallest group average were then taken to be representative of the typical ranges of *EMG* & ΔEMG values. TA: Tibialis Anterior; M-GA: Medial Gastrocnemius; VL: Vastus Lateralis; EMG: Electromyogram; *EMG*: EMG amplitude; ΔEMG : Relative change in EMG between pre-Vibration and Vibration normalised to the Vibration value; ISCI: Incomplete SCI; CSCI: Complete SCI; SCI: Spinal Cord Injury.

pBWS [%]	Muscle	<i>EMG</i> μV			ΔEMG [%]		
ISCI	TA	0.32	–	0.70	34.7	–	76.0
	M-GA	0.39	–	1.39	31.8	–	67.7
	VL	0.44	–	0.98	7.5	–	50.3
CSCI	TA	0.20	–	0.43	6.0	–	39.0
	M-GA	0.36	–	0.43	12.3	–	51.1
	VL	0.28	–	0.52	5.0	–	27.7

In summary, these results indicate that the M-GA was the most active muscle group during the Vibration epoch and the TA was the least active, with the VL in between for both SCI groups. Furthermore, the relative change in activity from the pre-Vibration epoch (baseline) to the Vibration epoch was largest for the M-GA muscle group and smallest for the TA, with the VL again in between in terms of ranking. Finally, while the relative change in neuromuscular activity of the ISCI were typically small they were always associated with an identifiable increase in underlying *EMG* activity, whereas some of the increase in activity for CSCI participants were drawn into speculation, because the absolute changes in *EMG* were very small and possibly random.

The main factors under investigation in this study were vibration frequency, amplitude,

muscle group, and classification of SCI. The neuromuscular responses during the Vibration epoch could not be attributed to any single one of these factors directly, but was rather a combined response to all of the parameters. However, general trends were observed which were consistent across the different muscle groups. It was confirmed that the initial conditions for each of the muscle groups were different (e.g. baseline level of neuromuscular activity prior to the application of vibration) and this likely had a significant effect on the type of response from each muscle group. It was concluded that the difference in response was primarily due to the relative proximity of the muscle to the vibration platform, whereby there was better transmission of vibration to the shank muscles compared to the thigh muscles. As a consequence the change in activity of the shank muscles was approximately 2.1:1 compared to approximately 1.6:1 for the thigh muscles. This was consistent with the absolute changes in neuromuscular activity. It was also noted that though these findings were in contrast to those for the neurologically intact population at 0% pBWS, they were consistent with the findings at 100% pBWS.

In summary, the relative changes in neuromuscular activity compared to baseline were large, but small in terms of absolute activity. It was thus concluded that the potential for hypertrophy or an increase in muscle performance using WBV-pBWS with an SCI patient population was small, however the application of this technique may have other potential therapeutic benefits such as the treatment of hypertonia, spasm and contracture. In addition, though the increases in neuromuscular activity were small, they could be used to augment existing functional capability in an ISCI group and be used to accelerate rehabilitation. Furthermore, the application of WBV may be used as an adjunct to proven rehabilitation technologies, such as FES, as it likely acts upon the neuromuscular system in a different manner and may potentially target different neuromuscular functional units.

EMG Vibration Cycle Analysis

The amplitude response analysis above was concerned with the steady-state neuromuscular response to WBV only, whereby averaging of the variability the EMG over a pre-determined epoch was carried out by computing the RMS. It was shown for the neurologically intact population in Chapter 5 that there may be a relationship between the neuromuscular response to WBV and a single cycle of vibration. Characteristic maxima in the EMG with a fixed latency were identified, possibly elicited during a period of peak platform acceleration.

The same analysis was repeated in this study. The EMG patterns were initially grouped according to the four main factors under investigation, which were: vibration frequency, vibration amplitude, muscle group, and classification of SCI. The EMG patterns were averaged according to each grouping factor and no characteristic maxima were identified for the pre-Vibration or post-Vibration epochs as expected. In contrast, a discernible maxima was identified for the Vibration epoch, despite not being as well defined as that for the neurologically intact population. This maxima was particularly discernible for the data grouped according to classification of SCI. The patterns for the other factors were not as clear, though a maxima related to vibration frequency was indicated.

A second analysis was carried out whereby the EMG recorded during the Vibration epoch only

were pooled according to muscle and SCI. For the acquired ‘EMG pattern’ to be classified as valid it also had to meet the following requirements: (i) the correlation between the ‘EMG pattern’ from the first and second testing session had to be statistically significant and (ii) the correlation had to be positive as determined using Pearson’s correlation coefficient. The criterion for a positive correlation was broad ($r \geq 0.1$) by necessity, due to the complexity and range of complications experienced during experimental testing with SCI participants. For reference, it is more common for an r -value of 0.7 or larger to be accepted as valid, however this threshold had to be reduced to 0.1 in this analysis because no single pair of EMG conformed to a correlation of 0.7 or greater. The r -value of 0.7 was previously determined to be excessively restrictive during the analysis of the neurologically intact population also.

No ‘EMG Pattern’ from the ISCI participants was retained in this second analysis, though a pattern was found for the CSCI participants. The TA and GA each exhibited characteristic maxima (or repeatable attributes) for the CSCI participants, though in the case of the TA the pattern did not fluctuate, and was akin to the pattern recorded for the pre- or post-Vibration epochs. The pattern from the GA was broadly similar to the results found for the neurologically intact population, where a maxima was present at 13% and 50% for the 12Hz and 20Hz vibration frequency conditions respectively.

It was concluded that though this relationship was shown, it was generally not as clear or consistent as it was in the neurologically intact population. The complexity of the case mix, which included additional co-factors such as classification on the AIS, level of injury and functional capability were likely to have precluded the ability to detect such changes in this sample.

Additionally, if the assumption that the maxima, which were observed for the neurologically intact population, were based upon reflex neuromuscular activity, as postulated by Ritzmann *et al.* [88] then it is likely that such phenomenon may never be observed for some SCI patient groups. Extensive disruption on normal central and peripheral neural function can exist depending on the level and severity of cord injury. Any component of a spinal reflex being induced by a WBV platform, should it exist, may never be manifested as a muscular contraction because the reflex signals may never reach the spinal cord, or be incorrectly interpreted by the spinal cord, or the neuromuscular system may have insufficient functional capacity to act upon the stimulus.

6.2.2 Technical

The technical objectives for this chapter were the same as those for the analysis of the neurologically intact participants. The objectives were principally to assess the transmission of vibration and then to determine the effect of the vibration on the EMG signal. The findings from the SCI participants and were in good agreement with the neurologically intact participants and were therefore not reported on extensively, but rather a succinct summary of the findings were reported instead.

There were no particular difference between the transmission of vibration to the neurologically intact and SCI participants, other than noting that vibration transmission decreased as pBWS

increased. This was most likely due to the reduction in ground reaction force, from the reduced load at the feet, and therefore less rigid contact between the feet and the platform. The most relevant result was the damping of vibration amplitude at higher frequencies in both the shank, and particularly the thigh. It is likely that the vibration amplitude was damped by the soft tissues, particularly the tendons, which acted like a low pass filter resulting in some absorption of the vibration energy, which was subsequently released as kinetic energy as the vibration switched direction. As a result, acceleration⁴ plateaued at approximately 4g in the shank and remained constant in the VL at approximately 1.5g.

Considering that acceleration was identified as central in determining the neuromuscular response during the vibration cycle analysis, and combining this with the fact that acceleration was stable across all combinations of frequency and amplitude applied at the feet, the constant neuromuscular output of the VL, rather than increasing, across all test conditions should be expected. By contrast the output of the TA and M-GA typically increased with platform acceleration as the shank muscles were affected by damping to a lesser degree.

Vibration

In summary, results show that vibration frequency was well correlated for all test conditions at all measurement locations. Acceleration and displacement measurements taken directly from the WBV platform closely match those expected. Acceleration and vibration amplitude of the foot increased with the vibration platform parameters but were not particularly well correlated with them. Meanwhile, a very small increase in acceleration and vibration amplitude of the VL was indicated for the ISCI participants, and the changes for the CSCI participants were negligible. The positive correlation between platform and foot vibration amplitude, and poor correlation between platform and VL vibration was expected and these same trends were indicated by the results from the neurologically intact participants.

These results are broadly similar to those from the neurologically intact participants where the same trends were identified. The results for all participants, including those without a SCI, are as follows:

- Vibration transmission was higher at lower levels of pBWS.
- Vibration was damped as it was transmitted away from the WBV platform and feet.
- Damping occurred in the form of an attenuation of vibration amplitude.

Damping may have occurred within the skeleton or soft tissue, though damping within the soft tissue was most likely. While indicated by these results, total damping could not be quantified with this data set, because all measurements were carried out at the skin, whose relative movement to the tissue underneath was unknown.

⁴Recall that acceleration is a function of frequency squared, and is linearly related to amplitude. Low pass filtering will therefore reduce the amplitude of vibration at that frequency and therefore reduce the detected acceleration overall.

It was concluded that the relative dose of vibration received by a muscle from the WBV platform may be inferred from the acceleration data, whose amplitude was attenuated by a reduction in vibration amplitude. Damping therefore acted to reduce the dose of vibration received by those muscle furthest from the vibration platform. It was therefore expected that the relative changes in the EMG due to vibration would be smallest in those muscles that were furthest from the platform. It was thus hypothesised that the excitation stimulus to the upper leg was predominantly moderated by vibration frequency because vibration amplitude had largely been damped. Whereas the vibration stimulus to the lower leg would have been equally moderated by vibration frequency and vibration amplitude. Finally, vibration artefacts in the EMG were expected to be present at the fundamental and harmonics of the vibration frequency, therefore simplifying the process of removing artefact from the EMG.

Artefact

As was found with the neurologically intact participants, artefact was found in every EMG recorded in this study. It typically comprised 19.4% of the EMG recorded from the ISCI participants, and 23.5% of the EMG recorded from the CSCI participants. However, during some particularly intense periods of vibration application, artefact comprised 94.3% and 97.5% of the total EMG power for the ISCI and CSCI participants respectively.

Statistically significant increases in the amplitude of the EMG were found for each of the first 8 harmonic frequencies in the EMG recorded from the ISCI participants. The overall effect size of these increases were medium-to-large for the first 5 harmonics only. The results for the EMG recorded from the CSCI participants were comparably better, where only the first 5 harmonic frequencies resulted in significant increases in the amplitude of the EMG, and the effect size of these increases were medium-to-large for the first 4 harmonics. The most relevant of these statistical results are presented in Table 6.15, as derived from the results presented in Figure 6.1.

Table 6.15: Summary of relevant statistical results from analysis of harmonics. n = harmonic number; $P_{total} = \sum_{i=1}^n h_i$ as a percentage of total EMG power; Effect Size (ES) is calculated between h_n and h_{n-1} ; ISCI = Incomplete SCI; CSCI = Motor Complete SCI; SCI = Spinal Cord Injury.

ISCI			CSCI		
Harmonic [n]	P_{total} [%]	ES	Harmonic [n]	$\%P_{total}$ [%]	ES
5	14.34%	0.111	4	18.16%	0.176
8	16.99%	0.029	5	19.86%	0.069
15	19.37%	0.009	15	23.49%	0.005

These results strongly indicate that vibration artefact resulted in an unacceptable addition to the amplitude of the EMG and should be removed offline using stop-band filters. To maintain consistency in the analysis of the data in Chapter 5 and considering the results above, it was concluded that it was adequate to remove the first 7 harmonics of vibration. A small, and in some cases statistically significant, percentage of artefact would have remained in the EMG, but the overall effect of the artefact on the results was deemed to be minimal.

Artefact content increased with vibration frequency and vibration amplitude for both the CSCI and ISCI participants. The EMG recorded during 12Hz or 1mm vibration typically comprised 12 - 16% artefact, compared to 16 - 24% artefact for the EMG recorded during 27Hz or 3mm vibration. Artefact content was typically larger in the EMG recorded from the shank muscles versus the thigh muscles. These results were broadly comparable to those from the neurologically intact participants. It was also noted that, in all cases, artefact content was highest for the CSCI participants.

These results confirm that EMG recorded during WBV contained a large and unavoidable proportion of artefact. Some have hypothesised that 'artefact' such as this has been misinterpreted, and is in fact genuine neuromuscular activity analogous to a short latency reflex. Furthermore, some supporting evidence of this hypothesis was presented in this work in the form of the vibration cycle analysis. The results however were inconclusive and it was determined prudent for this study to remove such artefact in order to avoid the potential overestimation of the EMG amplitude.

The results of the case study described in Section 6.1.3 strongly supports this standpoint. The study precluded the possibility of the observed maxima in the EMG as originating from spinal reflex activity, and confirmed the presence of vibration artefact in the EMG. It showed that the induced spikes in the EMG could be misinterpreted as genuine neuromuscular activity. When viewed in the frequency domain, the spikes were confirmed as being present in a flaccid muscle and therefore could not have originated from spinal reflexes. Furthermore, the spikes were effectively removed by filtering, thus reducing the amplitude of the EMG recorded during the Vibration epoch from the flaccid muscle to pre-Vibration levels. Similarly, the spikes were also found in a neurologically-intact muscle of the same participant, and while these spikes were reduced by filtering, it resulted in only a minor reduction in EMG amplitude.

Artefact signals were again limited to specific frequency bands which nominally occurred at the vibration frequency and its harmonics. This simplified the process for artefact removal. Results from the statistical analysis showed that it was adequate to specifically remove the first 5 harmonics of the vibration frequency, however, the first 7 harmonic frequencies were removed in this analysis to maintain consistency with the analysis carried out for the neurologically intact participants.

EMG Classification

Vibration artefact was not the only source of error found in the EMG recorded for this study. It was observed during testing that WBV would elicit a series of clonic spasms in some of the SCI participants, and while this phenomenon is of interest, it was not under investigation in this study. It was therefore deemed that the corresponding EMG showing this spasmodic neuromuscular activity be removed from the analysis.

The procedure for rejecting these EMG was automated in order to maintain consistency and remove the element of 'human error'. The same custom algorithm, as that used previously, was used here. The ratio between the burst-like (spasm) to tonic activity was used to determine whether the EMG contained outlying data. 680 EMG were ultimately rejected

as outliers. This accounted for 15% of all recorded EMG, 43% of which were recorded during the post-Vibration epoch which was not considered in the subsequent analysis in any case. These rejection rates were considerably higher than those rejected from the neurologically intact participants and the majority of the rejected EMG were from the shank muscles. There was also an increase in the number of rejected EMG with increasing vibration frequency and amplitude. These results were broadly comparable to those from the neurologically intact population. Most of the rejected EMG were from the ISCI participants. This was expected because those subject with an incomplete injury typically had stronger muscles, therefore when a spasm did occur it was more likely to be detected by the EMG electrodes and rejected by the algorithm.

BTR statistics of the accepted EMG were calculated. There was no statistically significant difference between tests, and the BTR was higher during the Vibration epoch than during the pre-Vibration epoch, and higher for the ISCI participants, as expected. It increased with vibration frequency and amplitude, and was approximately the same for the different muscle groups.

These results confirmed that the application of WBV with pBWS was feasible in a SCI population. Despite the comparably large number of rejected EMG, over 3,800 discretised EMG were retained for further analysis. The BTR values corroborated the physiological findings above which confirmed an increased neuromuscular response during the application of WBV.

6.3 Conclusions

It was shown that WBV-pBWS was feasible in both neurologically intact and SCI populations. This was confirmed by the increased neuromuscular response found during the Vibration epoch. Furthermore, increases in neuromuscular activity were identified with all levels of pBWS, including 100% pBWS when applied with a neurologically intact population. It was shown that pBWS may be used to facilitate conventional WBV training (i.e. with the participant standing on the vibration platform). The same measurements and analysis techniques were applied during the study of SCI and neurologically intact participants allowing for the results of each group to be directly compared with one another.

Overall, the neuromuscular responses to WBV were small, irrespective of vibration frequency, vibration amplitude, muscle group, level of pBWS or classification of SCI. The increases in neuromuscular activity were typically less than 10% MVC for the neurologically intact participants and the likelihood of muscle hypertrophy occurring in a SCI population as a result of WBV training was concluded to be small, particularly because a SCI population would typically carry the WBV exercises with higher levels of pBWS.

Acceleration measurements indicated that the vibration was damped as it was transmitted throughout the legs, and that it was damped to the same degree regardless of a SCI or not. The magnitude of the damping decreased with the proximity of the muscle to the vibration platform. Vibration transmission was also reduced with increasing levels of pBWS. It was

thus concluded that the dose of vibration was lowest for the neurologically intact population at 100% pBWS and CSCI participants.

Damping reduces the total dose of vibration received at a particular location and therefore directly impacts upon the magnitude of the neuromuscular response. This is evidenced by the fact that the relative changes in EMG (ΔEMG) were consistently larger for the shank muscles than the thigh muscles. However, despite the relative change in activity recorded, the absolute magnitude of neuromuscular activity achieved by the muscle during vibration (EMG & EMG_N) was primarily predicated on the level of contraction in the muscle prior to the onset of vibration. This is evidenced by the fact that the most active muscles both before or during vibration were the GA and VL, regardless of neurological injury or not. However, in all cases the magnitude of the response to WBV was small, despite the fact that the relative change to baseline was often large.

It is difficult to directly compare the absolute levels of activity for the SCI participants to those from the neurologically intact population, because the EMG from the SCI participants were not normalised to a MVC. Despite this limitation, it was concluded that the absolute level of activity achieved by the SCI participants was small. There are a number of indicators for this conclusion: (i) the ΔEMG for the SCI participants and those from the neurologically intact population were similar. Therefore, since it is known that the EMG_N values for the neurologically intact population are small, it can thus be inferred that the values for the SCI participants are small also; (ii) The EMG values recorded from the SCI participants were less than $16\mu\text{V}$, and while these values may not be used for inter-subject comparisons, they are generally considered to be small; and (iii) ΔEMG are similar for both test populations, however the SCI participants are most likely starting from a much lower baseline due to disuse induced muscle atrophy, a large ΔEMG would still be equivalent to a small absolute level of muscle activity.

The presence of vibration artefact was initially described during the analysis of the results of results from the neurologically intact participants (Chapter 5) where it was concluded that artefact significantly increases the amplitude of the EMG. It was shown that the artefact is effectively eliminated by using notch-filters specifically at the frequency of vibration and its harmonics, and that it is sufficient to remove to first 7 harmonics only. Supporting data from the case study carried out with a SCI participant confirmed that the ‘spikes’ in the power spectrum of the EMG were more likely to be vibration artefacts rather than a short latency reflex. The outcome of the case study was twofold: (i) it was known that a neuromuscular response cannot be elicited in a flaccid muscle, therefore the increase in the amplitude of the EMG were confirmed to be artefact, and (ii) applying notch-filters to the first 7 harmonics of vibration effectively reduced the magnitude of the EMG to pre-Vibration levels, and were therefore effective in eliminating artefact from the EMG.

Clear trends were identified from this small and complex case mix. This cross-sectional study recruited early-stage SCI participants of differing levels of cord injury, severities of injury, time since injury, and possible bi-lateral limb imbalances. Further analysis of the data grouped according to AIS classification and ASIA muscle scores could perhaps elucidate

further a particular ISCI or CSCI subgroup that may benefit from WBV training to a greater degree, however insufficient data was collected in this study to carry out such an analysis. The results for the SCI participants were similar to those results for those from the neurologically intact population when they were provided with 100% pBWS. Vibration caused a small but confirmed increase in neuromuscular activity, and the magnitude of the increase was determined to be elicited by peak platform acceleration. It was concluded that despite the increases being small, WBV-pBWS should be investigated further as a rehabilitation technology. In the case of the CSCI participants, any increase in neuromuscular activity is larger than the maximum voluntary capacity of the muscle and is therefore of potential benefit. In the case of the ISCI participants, the WBV platform may be used to augment existing functional capability and could be employed as an adjunct to existing muscle strengthening or rehabilitation exercises.

Chapter 7

Conclusions & Future Work

A cycle of decline in cardiovascular and pulmonary health of early-stage SCI patients, accompanied by muscle atrophy, bone demineralisation and orthostatic intolerance were highlighted at the beginning of this thesis. The scope of the work set out was to focus on rehabilitation technologies which may be employed during early-stages SCI rehabilitation and used to attenuate some of these declines.

Two suitable technologies were identified: RATTT and WBV-pBWS. In the case of RATTT, the commercial system was sufficiently well developed such that it was adequate to integrate a cognitive feedback system to encourage volitional participation, and then measure cardiopulmonary and vascular output during robotic-assisted stepping, and robotic-assisted stepping with the support of FES. In the case of WBV, the neuromuscular response of a SCI population to vibration was poorly understood with only limited evidence available in the literature. Furthermore, an adequate system to test WBV with a neurologically compromised population did not exist. A WBV platform was therefore integrated with a pBWS system to facilitate testing in this population.

Having identified two such technologies for application in SCI, the following objectives were set out: (i) determine the feasibility of RATTT as a form of exercise during early-stage SCI by measuring the acute vascular, pulmonary and ventilatory responses of an early-stage SCI patient population during RATTT training and assess if the training could be of sufficient intensity to potentially attenuate the decline in fitness, and (ii) develop a rehabilitation system that allows those with an early-stage SCI to stand on a WBV platform, measure and quantify the magnitude of the acute neuromuscular response of a neurologically intact and SCI population to WBV in order to determine the feasibility of the system for early-stage SCI rehabilitation, and then assess if the response was of sufficient magnitude to attenuate or counteract muscle atrophy.

7.1 Conclusions

In the case of both RATTT and WBV, the initial requirement was to assess the feasibility of each and determine if they could be applied for SCI rehabilitation. One of the principal contributions of this thesis was the demonstration of this feasibility. RATTT and WBV were well tolerated by the neurologically intact and SCI populations, and no severe adverse

effects were reported. In both cases there was concern of orthostatic instability arising due to the verticalisation of a patient group unused to being in a standing position. RATTT was identified as having the smallest risk of this occurring, due to the activation of the skeletal muscle pump during robotic stepping, and the fact that patients were verticalised to 70° from supine rather than being fully upright. In the case of WBV-pBWS, there was one instance of pre-syncope during testing, as is possible during any form upright training with SCI patients. There were no other adverse effects which were reported in the literature, such as erythema.

In addition to the successful trial of these systems with SCI patients, both were shown to have potential for a positive effect. This was particularly the case for the ISCI participants who exhibited increased metabolic demand and heart rate during robotic stepping and relatively large changes in neuromuscular activity during WBV. The corresponding changes from the CSCI participants were typically not as large, but some increases occurred nonetheless. In the case of both technologies, it was possible for the ISCI participants to utilise their remaining function to augment the effect of the rehabilitation technology. In this regard, the feasibility of both technologies was demonstrated. Not mentioned in either study was the potential psychological benefit of carrying out rehabilitation in an upright position. This was an aspect of the technologies that was frequently remarked upon during testing but was not under particular investigation in this thesis and as such was not discussed any further.

The second aspect of the RATTT investigation was to assess if the training was of sufficient intensity to attenuate the decline in fitness. The metabolic demand of the ISCI participants was equated to 1.5 - 3.1 METs, which is considered to be of low to moderate intensity by the WHO. The metabolic demand of the CSCI participants was equated to 1.4 - 2.1 MET's, or low intensity activity. In both cases though, these levels of activity were typical of the maximum achievable metabolic demand in the absence of any training of the SCI patients. It was thus determined that RATTT was suitable for SCI rehabilitation. Furthermore, it was noted that the intended application of this technology was for early-stage SCI rehabilitation only. It was shown that it can be applied with success, and no adverse effect or instance of OH occurred, thus resulting in an increased metabolic demand. It is proposed that RATTT is used in this way until such a time that the patient is comfortable with being in an upright posture and can then progress to other forms of proven exercises such as walking (if functionality permits) or cycling, with or without the support of FES.

The second aspect of the WBV-pBWS investigation was to describe the steady-state neuromuscular response during its application, principally because little or no evidence existed in the literature to that effect. A limited number of studies investigated the effect of WBV on changes in isokinetic strength and blood flow in neurologically compromised patient groups, with little supporting evidence available on which vibration conditions should be applied [115,136]. The results from Chapters 5 & 6 confirmed that WBV-pBWS may be used to elicit an increase in neuromuscular activity and it was shown that increasing frequency or amplitude elicited an increased neuromuscular response. Each of these parameters were combined into an acceleration value, and the vibration cycle analysis indicated that it was platform acceleration that ultimately moderated the neuromuscular response. This was the second principal contribution of this thesis: providing a systematic and detailed description of

the steady-state neuromuscular response to a range of low, medium and high WBV platform accelerations.

Confirmation that acceleration moderated neuromuscular output was evidenced further by the fact that the neuromuscular activity of the shank muscles typically increased with acceleration regardless of neurological injury or not, meanwhile, acceleration data indicated that the transmission of vibration to the thigh was low due to the attenuation of vibration amplitude, resulting in a non-specific increase in VL neuromuscular activity during WBV. It was thus concluded that in order to achieve training of the shank muscles with WBV, it would be adequate to increase frequency or amplitude, however a corresponding increase in output of the thigh muscles should not be expected. Overall, the magnitude of the response to WBV-pBWS were relatively small, thus precluding the likely effectiveness of WBV-pBWS for improving muscle strength or power in SCI. However, other potential applications of WBV for rehabilitation in SCI were identified.

7.2 Future Work

7.2.1 Limitations & Alternative WBV Support Systems

One of the principal challenges in applying WBV-pBWS in SCI was fitting the participant with the support harness, and achieving an appropriate standing position. This was particularly difficult with the CSCI participants who in some cases also exhibited hypertonia of the hip flexors and extensors. In these cases it took considerable time to get the leg to stretch and settle into the prescribed position. In some cases, it was preferred if the participant had spent time stretching in a standing frame prior to taking part in an experimental session. This was typically part of a standard rehabilitation regime in the spinal unit though it was not a requirement for inclusion in this study.

Secondly, in some cases the feet of the SCI participants would move during testing with high platform accelerations, particularly with the CSCI participants. While this movement was not reflected in the acceleration data, it was concluded that the foot was able to move because there was insufficient ground reaction force, and therefore insufficient friction to hold the foot in place.

It is therefore proposed that it may also be appropriate to integrate a WBV platform into a standing frame. Full support would be provided by the frame while maintaining a large component of body weight being supported by the legs. There would therefore still be a large ground reaction force. In many cases, a standing frame is more practicable than a body weight support system, and may therefore be more likely to be used in a clinical environment. Increased loads could be achieved with this system by increasing frequency or amplitude. A limitation of the application of vibration in this way however is the inability to control the load carried by the participant themselves. In a worst case scenario the participant could simply allow the frame to take all of the body weight. There would consequently not be a prior level of contraction in the muscle, which is important in modulating the magnitude of response achieved during vibration. The advantage of the pBWS system in this regard is that the participant must use some volitional effort where appropriate.

To overcome this limitation with a standing frame, a force transducer could be built into the base of the standing frame. Feedback could be provided from the force transducer indicating how much load was being carried by the standing frame and how much load was being carried by the participant. The integration of a force transducer would also open up the possibility of comparing the neuromuscular response to different levels of ground reaction force.

7.2.2 Muscle Performance

The work in this thesis described the acute neuromuscular responses of neurologically intact and early-stage SCI volunteers to WBV-pBWS. The next logical step is to investigate the effect of a longer term regular WBV training programme on muscle strength. Despite the conclusion that the overall increase in neuromuscular activity during the vibration was small, it was generally larger than that which could be achieved by the SCI participant on their own. Furthermore, WBV typically resulted in larger gains in untrained populations (Section 3.6) and empirical evidence generated as part of the EMG studies presented in this thesis provide justification of which vibration parameters should be used and which muscle groups tested.

It is thus proposed that the effectiveness of a longitudinal WBV training programme be assessed in a chronic SCI (>1 year since injury) patient population. It is important that the effectiveness of the training programme be compared against an established exercise programme, such as recumbent FES cycling, as this has been a major shortfall in assessing the effectiveness of other WBV training programmes reported in the literature. Ideally the investigation should be a randomised control trial, with patients randomly allocated to the WBV or standard group. A chronic SCI patient group should be selected as their functional capability would have plateaued and therefore any change in muscle performance could be more reliably attributed to the intervention. The drawback of this approach is that the nerves and muscle tissue may have degraded to such a point that the training period required would be extensive and the gains may be minimal, since the level of muscle performance would likely be very low to begin with.

An increase in training load should be achieved with the ISCI patients by first decreasing the level of pBWS and then by increasing platform acceleration. The VL and M-GA were typically found to exhibit the largest neuromuscular response during vibration, and as such a pre-contraction of the muscle should be encouraged by performing heel lifts and deep squats. Dynamometry could be used at regular intervals to assess changes in muscle strength.

Increased training load with the CSCI patients may only be achieved by increasing platform acceleration. Effective transmission of the vibration to the CSCI patients legs is key, due to the increased tendency of their legs to move during instances of high platform acceleration. Strapping of the feet to the WBV platform would be one such approach. FES should be used with dynamometry to assess any changes in muscle strength.

7.2.3 Contraction Onset Latency

This study characterised the steady-state neuromuscular response to different WBV conditions, and it was assumed that steady-state had been achieved within the 30s time frame during which vibration was applied. Another potential aspect of the neuromuscular response to WBV would be to investigate the onset latency of the contraction during WBV. The question arises: is the neuromuscular response immediate and constant soon after the application of vibration? Or does the neuromuscular response start slowly and build gradually as it does with a TVR?

The same experimental methodology as was used in this thesis could be used again. However, it is proposed that a single vibration condition be used, apply vibration for a longer period of time, repeat in triplicate, and investigate in a larger test population. During the analysis, the RMS should be calculated with a relatively small moving window such that the onset of the contraction is not averaged out if it occurs quite rapidly.

7.2.4 Spasticity

Spasm is a major contributing factor which inhibits the ability to participate in effective forms of rehabilitation. Results from this study indicate that WBV may provide sufficient stimulation to modulate the neural pathway which leads to spasm and therefore potentially act to reduce it, while at the same time not significantly acting to increase muscle strength or power. The use of WBV to treat spasticity was initially proposed by Ness and Field-Fote [173]. Clonus and hypertonia, which were both a consequence of hyperexcitability of the α -motoneuron among other complicating factors, resulted in an increase in muscle tone or contraction from a very minor stimulus.

WBV may be particularly suited to the treatment of spasticity for two main reasons. Firstly, it is apparent that WBV increases neuromuscular activity which primarily acts via the α -motoneuron. It is hypothesised that repeated stimulation of the α -motoneuron by WBV could ultimately lead to an increase in the de-polarisation threshold of the α -motoneuron and therefore decrease in its excitability. The excitability of the α -motoneuron could be assessed by measuring the H-reflex at repeated intervals during a longitudinal course of WBV treatment. Secondly, the intention of any treatment of spasm would be to ensure that the treatment itself would not make the spasm worse, for example by increasing muscle strength, in which case the consequence of the spasm would be considerably more severe. The findings from this study show that the likelihood of any increase in muscle strength from WBV applied with pBWS is small.

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Appendices

Introduction To Neurophysiology

The Nervous System

The human nervous system comprises the central and peripheral nervous systems. The central nervous system consists of the brain and spinal cord, and the peripheral nervous system consists of cranial and spinal nerves. The functionality of the nervous system is divided into two subcategories: the autonomic, and somatic nervous systems. The somatic nervous system allows volitional movement to be achieved through interaction with the peripheral nervous system. The peripheral nervous system conveys effector signals away from the central nervous system towards muscles and glands (efferents), and conveys sensory information from sense organs towards the central nervous system (afferents). Afferents, and efferents, are volleys of action potentials which are generated and transmitted by nerve cells. As such, the nerve cell is considered to be one of the most basic functional units within the nervous system.

Spinal Cord

The spinal cord connects the brain to the peripheral nervous system and is made up of white and grey matter. It extends caudally from the base of the skull to the conus medullaris, which is located approximately at the L1\|L2 vertebral level [181]. The function of the spinal cord is to transport sensory and motor information to and from the brain. The peripheral region of the spinal cord contains longitudinally oriented, ascending and descending, sensory and motor tracts [8]. The myelin sheath(s) that protect the axons of these ascending and descending tracts are white in colour, and the region is so called white matter. Contained within the white matter is a butterfly shaped region called grey matter, which is a densely packed region of neuronal cell bodies. The spinal cord is housed within the bony vertebral column, which is considered as four separate and distinct regions, known as the cervical, thoracic, lumbar and the sacral regions. The cervical, thoracic and lumbar regions are made up of 24 articulating vertebrae and the sacral region is fused.

The spinal cord is involved in many bodily functions from pupil dilation and sweating to movement, and its functional activity is carried out through the somatic and autonomic nervous systems. The somatic nervous system innervates skeletal muscle, and is generally involved in coordinated muscle activities for posture, movement and control. The autonomic nervous system innervates smooth and cardiac muscles, and glands, and is involved in involuntary bodily functions. In some cases the somatic and autonomic nervous systems can and do interact, for example in the maintenance of body temperature. If there is a drop in body temperature, the somatic nervous system initiates heat generation by eliciting a contraction in skeletal muscle, while at the same time the autonomic nervous system minimises heat loss by eliciting vasoconstriction [181].

Autonomic Nervous System

The autonomic, or visceral, nervous system is composed of three subdivisions, which include: (i) the sympathetic, (ii) parasympathetic, and (iii) eccentric divisions. Sympathetic, or thoracolumbar, outflow occurs between spinal segments T1 - L2. It is generally involved in 'fight-or-flight' responses and innervates the heart and lungs, abdominal viscera, and urinal, genital and lower digestive systems [181]. Parasympathetic, or craniosacral, outflow occurs from nerves that take their origin from the cranium and spinal segments S2 - S4. It is

generally involved in conservation and restoration of resources, and is mainly involved in urination, defecation, and erection mechanisms [181]. The eccentric division is involved in the functioning of the gut, and is described as the ‘gut-brain’ as it can operate without central nervous system control [181].

Sympathetic and parasympathetic outflow both exert control over certain systems such as the heart, lungs and gastrointestinal tract. Sympathetic function can elicit an increase in heart rate, dilation of blood vessels and bronchial lumen and stimulation of the sphincter, whereas the parasympathetic system will decrease heart rate, constrict bronchial lumen and inhibit the sphincter.

Somatic Nervous System

The somatic nervous system communicates with higher level cortical centers, the peripheral nervous system, and the autonomic nervous system, via interneurons and spinal roots. The somatic nervous system is responsible for the control of all volitional motor activity and innervation is taken from all spinal segmental levels. The motor and sensory signals involved in voluntary movement are conveyed via spinal roots, and innervation of the roots for a particular part of the body is generally taken from one segmental level. During volitional motor tasks, an efferent is sent from the central nervous system, via the peripheral nervous system to a muscle cell resulting in a contraction. This contraction is detected by sense organs which send a sensory signal back to the central nervous system, therefore creating a closed loop system and permitting control over the motor output.

Involuntary motor activity is not part of the somatic nervous system and occurs where a sensory nerve synapses directly with a motor nerve cell. This therefore results in a short circuit of the path to the muscle cell, and bypasses the central nervous system. Such involuntary activity is described as a spinal reflex, whose function is generally to protect. Spinal reflexes are characterised by their short latency and examples include the withdrawal and myotatic reflexes.

Spinal Roots

Dorsal and ventral roots, which carry sensory and motor information respectively, combine to form a spinal root (Figure 1). Sensory afferents are conveyed towards the posterior horn of the spinal cord from discrete regions of the peripheral nervous system known as dermatomes. Motor efferents from the anterior horn of the spinal cord are conveyed to a group of muscle fibers, known as a myotome. It is possible for a root to innervate more than one muscle and one muscle to be innervated by more than one root [8].

The spinal roots, of which there are 31 pairs, are named based on the vertebral foramina through which they pass. There are 8 pairs of cervical roots, 12 pairs of thoracic roots, 5 pairs of lumbar roots, 5 pairs of sacral roots and a single coccygeal root [182]. The head, neck, arms and hands are supplied by the roots that take their innervation from the cervical spine (C1 - C8). The chest and trunk are supplied by the roots innervated at the thoracic level (T1 - T12). The legs are innervated at the lumbar level (L1 - L5), and the feet and anus

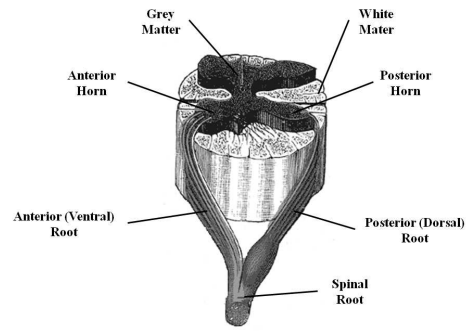


Figure 1: Schematic of spinal segment with spinal roots (adapted from Grays [175]).

are innervated at the sacral level (S1 - S5). Since the spinal cord is shorter than the vertebral spinal column itself, the lumbar and sacral roots are longer and extend caudally within the spinal cistern known as the cauda equina (horses tail) [181]. A basic schematic is outlined in Figure 2 identifying the relationship between different regions of the body and the spinal column.

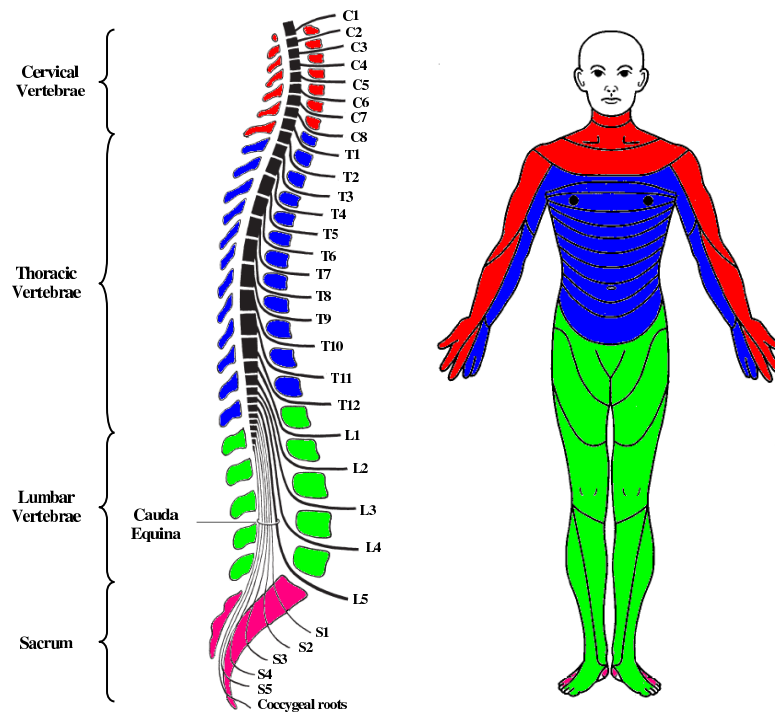


Figure 2: Schematic of spinal column and cord with corresponding dermatome and myotome regions (adapted from Noback *et. al.* [181]).

Volitional movement occurs when an action potential is generated by the somatic nervous system and propagated along the relevant spinal root to the appropriate neuromuscular junction, eliciting a MUAP. As the MUAP propagates along the muscle, it elicits a contraction of the individual muscle fibers within the muscle.

Muscle Fiber

The extrafusal muscle fiber is one of the most basic functional units that comprises striated muscle. The basic components that make up a muscle fiber are outlined in Figure 3. The contraction of a muscle cell is described by the sliding filament theory.

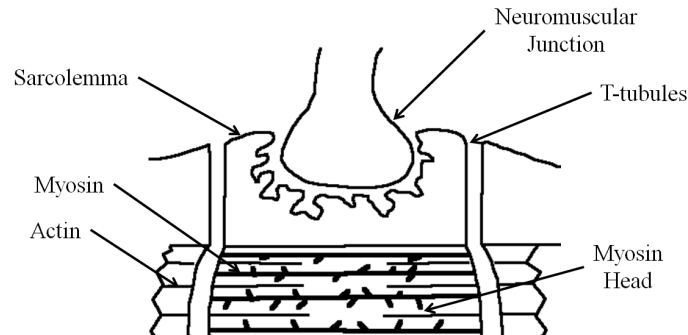


Figure 3: Basic schematic of a muscle fiber describing the main components involved in the contraction of muscle (adapted from Branstrom *et al.* [183]). Not shown in this figure are troponin, tropomyosin, the terminal cisterna, mitochondria, the binding sites on actin for the myosin heads, or the binding sites on the myosin heads for the adenosine triphosphate molecule. During a muscle contraction, the actin subunits are drawn together by multiple cross bridging cycles between actin and the myosin heads therefore causing the muscle to shorten.

Single Cross Bridge Cycle

During a single cross bridge cycle, a MUAP propagates along the sarcolemma and down the t-tubules of a muscle fiber, stimulating the release of Ca^{2+} from the terminal cisternae (not shown). As the Ca^{2+} ions enter the intracellular fluid (cytosol), they bind to troponin, therefore changing the troponin-tropomyosin complex conformity on the actin subunit. This change in conformation exposes the myosin binding sites on the actin subunit. Energised myosin heads, found on the myosin chains bind with the exposed binding sites, forming a cross-bridge. Once bound, the myosin head releases Adenosine Diphosphate (ADP) and inorganic phosphate, causing the myosin head to flex and pull the actin in. This is called the power stroke. At the end of the power stroke, an ATP molecule binds to the myosin head, and is converted into ADP and inorganic phosphate in a process called hydrolysis, therefore energising the head and causing it to disconnect from the actin subunit. The Ca^{2+} ions are pumped back into the terminal cisternae, restoring the troponin-tropomyosin complex conformation, and covering the myosin binding sites. A sustained contraction of muscle occurs during multiple cross bridge cycling.

ATP is a key component of the single cross bridge cycle and it is produced by the mitochondrion. ATP is used during the power stroke and during the pumping of Ca^{2+} in and out of the terminal cisternae. A small store of ATP is available within the muscle, however during periods of sustained activity it is necessary to generate additional ATP. Mitochondria are therefore commonly described as the ‘cellular power plant’.

ATP Production

ATP is produced by one, or a combination, of the following processes: (i) hydrolysis of creatine phosphate, (ii) glycolysis, or (iii) oxidative phosphorylation. Hydrolysis of creatine phosphate and glycolysis can occur in the absence of oxygen and are therefore described as anaerobic. Oxidative phosphorylation occurs with oxygen and is therefore described as aerobic.

During rest and recovery, lactic acid built up in the muscle is converted back into pyruvic acid, which is converted into CoA which is used to produce ATP, carbon dioxide, and water. ATP is then used to replenish creatine phosphate stores from creatine.

Fiber Type

Muscle fibers are broadly categorised into three sub-groups based on their physical, electrical, and axonal properties: (i) slow twitch (Type I), (ii) fast twitch-fatigue resistant (Type IIa), and (iii) fast twitch (Type IIb). The general properties of these muscle fibers are described in the Table 1. The fatigue resistance of a muscle fiber is related to the capillarisation, and number of mitochondria present in the fiber. A fiber is more resistant to fatigue if it is oxidative, i.e. a plentiful supply oxygen is available, and it can readily produce ATP due to a large number of mitochondria. In the absence of oxygen and mitochondria, a muscle fiber will be more reliant on glycolytic, or anaerobic, processes and it will therefore be more readily fatiguable.

Table 1: Summary of the mechanical, electrical, and axonal properties of the different types of muscle fiber.

Fiber Type	Mechanical	Electrical	Axon
Type I	Slow twitch; small force production; fatigue resistant; small fiber diameter	Slow nerve conduction velocity	Small; recruited for low force production
Type IIa	Fast twitch; fatigue resistant	Intermediate nerve conduction velocity	Intermediate; recruited for moderate force production
Type IIb	Fast twitch; high force production; fatiguable; large fiber diameter	High nerve conduction velocity	Large; recruited for high force production

Muscle activity can be studied by inferring the chemical and electrical processes that are occurring during a muscle contraction. The MUAP which elicits a cross-bridge cycle resulting in a muscle contraction can be measured using EMG. The chemical processes within the mitochondria leading to the generation of ATP which is used as a fuel during a muscle contraction can be monitored by measuring gas exchange at the mouth. These techniques are discussed in greater detail in Section 1.1.

Sensory Receptors

While movement is achieved through the contraction muscle fibers, the motor output is controlled by the central nervous system based on feedback of the movement from sensory receptors. Two examples of sensory receptors that are found in muscle are muscle spindles and Golgi tendon organs. These receptors are integral to proprioception and control of movement.

Furthermore, they are thought to play a role in the neuromuscular response to WBV which is discussed in greater detail in Chapter 3.

Muscle Spindle

The muscle spindle is a sensory receptor that lies in parallel with the extrafusal muscle fibres of skeletal muscle. Its function is to detect the change in length of skeletal muscle, and it is thought to play a central role in the neuromuscular response to WBV. The muscle spindle is encapsulated with modified muscle fibres called intrafusal fibres. The intrafusal fibres moderate the sensitivity of the muscle spindle during both static and dynamic contractions of skeletal muscle. The spindle thus has both sensory and motor innervation.

There are two sensory fibres that innervate the muscle spindle: type Ia and type II fibres. The type Ia fibre plays a central role in the stretch reflex. Motor innervation is provided via the γ -motoneuron, which itself is subdivided into γ -dynamic and γ -static motoneurone. The γ -motoneuron innervates the intrafusal fibres, causing them to contract and increase the sensitivity of the muscle spindle. The γ -dynamic motoneuron is essential to allow the body to react to external disturbances while the γ -static motoneuron ensures that the spindle remains taut when the extrafusal fibres are held at a constant length.

The Ia fibre synapses directly with: the α -motoneuron that supplies the same muscle (homonymous muscle), the α -motoneuron that innervates synergistic muscles, and interneurons that inhibit antagonistic muscles (heteronymous muscles). Type II fibres can also synapse directly with the α -motoneurone innervating homonymous muscles.

Golgi Tendon Organ

The golgi tendon organ is found at the tendinous insertion of the muscle and it detects tension. It is innervated by type Ib sensory fibres that act to inhibit homonymous muscle activation. Chief among its functions are to prevent damage to the tendon from excessive forces generated by the muscle and to facilitate contraction of the antagonistic muscle through reciprocal inhibition.

Spinal Reflexes

The command to elicit a contraction in skeletal muscle, and therefore movement, normally originates from the central nervous system. Central commands via the somatic nervous system are normally voluntary. In some instance, commands originating from the peripheral nervous system can also elicit a contraction in muscle. These are called spinal reflexes, and they are involuntary, have a short latency, and they bypass the central nervous system. A simple example is the stretch, or myotatic, reflex.

A stretch reflex of the quadriceps muscle group (knee jerk reflex) can easily be achieved by tapping the patellar tendon with a tendon hammer. Striking the patellar tendon causes it to shorten, resulting in a stretch of the quadriceps. This is detected by the muscle spindle, which sends a volley of action potentials via the Ia fibre, to synapse with the homonymous α -motoneuron. These action potentials are excitatory and act to depolarise the α -motoneuron.

and cause the muscle to contract, thereby shortening back to its original length. For this reason the stretch reflex is described as monosynaptic (see Figure 4), i.e. it involves only one synapse.

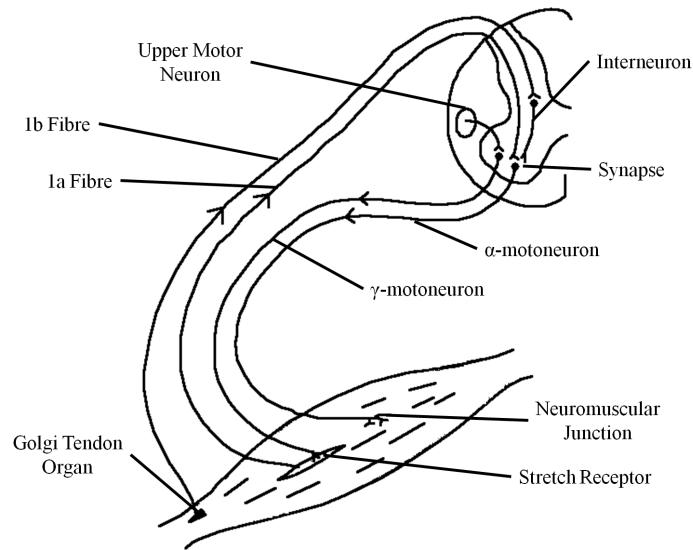


Figure 4: Schematic of a stretch reflex (adapted from Noback *et al.* [181]).

In addition to synapsing with the homonymous α -motoneuron, the 1a fibre also synapses with interneurons that inhibit α -motoneuron activity in the antagonistic muscle. In this example the quadriceps will thus contract and the hamstring will relax, causing the knee to jerk. This highlights the interaction between motor-, sensory- and interneurons, and the role they each play in the generation of human movement. It has been widely suggested in the literature that the stretch reflex plays a central role in the neuromuscular response to WBV (Chapter 3).

The Jendrassik maneuver may be used to potentiate the stretch reflex. In this maneuver, the subject is asked to interlock their fingers and pull. Facilitatory action potentials are thought to descend from higher levels and potentiate the response to stretch. The H-reflex is analogous to the stretch reflex, however instead of stretching the muscle to elicit the contractile response, an electrical stimulus applied to the spinal root, thereby directly depolarising the α -motoneuron, and eliciting a contraction in the homonymous muscle group.

To better understand reflexes, and the interaction between sensory fibers and muscle cells it is necessary to describe the nerve cell in greater detail.

The Nerve Cell

Efferent nerve cells are described as UMN if located in the central nervous system, or LMN if located in the peripheral nervous system. Most UMN are found within the white matter of the spinal cord. The majority of nerve cells found within the grey matter of the spinal cord are interneurons, which interconnect the ascending and descending spinal tracts, and the peripheral nervous system.

The nerve cell itself consists of a cell body (soma), a dendrite and an axon. The axon, which is protected by an insulating sheath called myelin (Figure 5), can have a number branches called collaterals. It stems from the axon hillock, and is the point at which an action potential is generated. An action potential is a small electrical impulses that allows all communication within the nervous system.

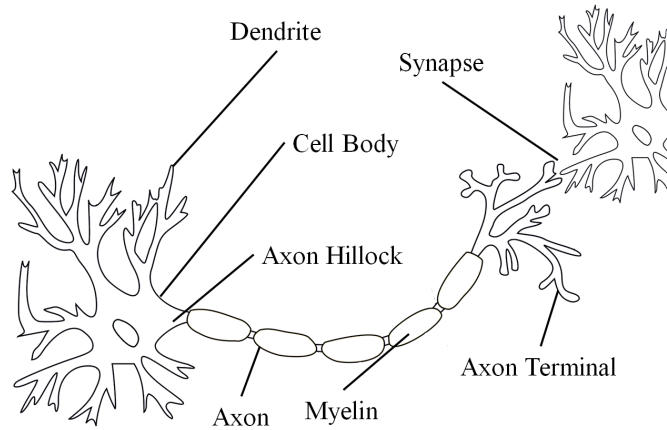


Figure 5: Schematic of nerve cell (adapted from Jaroz [184]).

Action Potential

An action potential is a large change the resting membrane potential of a nerve cell (Figure 6) that results in a change in the permeability of the cell to Na^+ and K^+ ions. The action potential is a characteristic of the nerve cell that generated it as it is always of the same magnitude. The action potential is generated at the axon hillock, where the density of Na^+ ions is greatest. The typical membrane potential of a nerve cell at rest is approximately -70mV , so called the resting membrane potential. If incoming signals from the dendrites and cell body are sufficient to bring the membrane potential to a threshold of approximately -55mV , voltage-sensitive Na^+ gates open, thereby changing the cells permeability to the Na^+ ions. The influx of Na^+ down their concentration gradient and into the cell results in a rapid increase in the membrane potential, and forms a positive feedback loop that opens additional voltage-sensitive Na^+ gates. The membrane potential will continue to rise to a peak value of approximately $+30\text{mV}$. This process is called depolarisation. If the initial stimulus is not sufficient to reach threshold, the voltage-sensitive Na^+ gates will not open and no action potential will be generated. It is for this reason that an action potential is described as an all-or-none event.

Two processes interrupt the positive feedback loop and bring the generation of the action potential to an end. The open Na^+ channels become inactivated by a time-sensitive gate that closes after a period of time, preventing an influx of additional Na^+ . Voltage-sensitive K^+ channels also become active. The K^+ channels respond slowly to the initial change in the membrane potential and only become fully active after the peak of the action potential has occurred. K^+ begins to move out of the cell, resulting in a decrease in the membrane potential back towards its resting value. This process is known as repolarisation. Due to the slow

response of the K^+ channels, there is a delay between reaching the resting membrane potential and the channels finally closing. This results in the cell membrane becoming hyperpolarised.

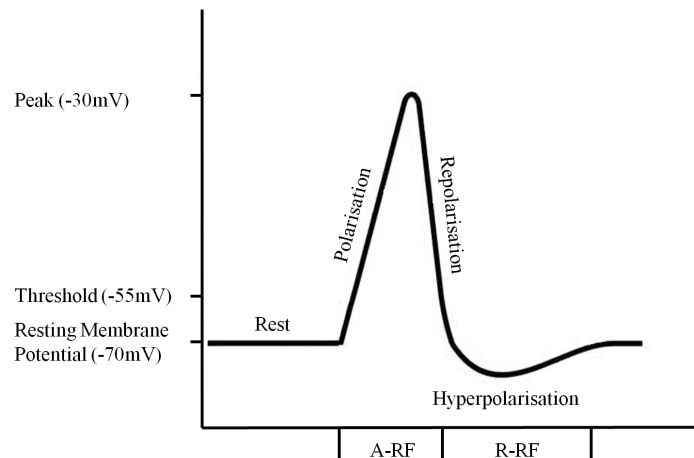


Figure 6: Schematic of action potential. A-RF: Absolute refractory period. R-RF: Relative refractory period

During the absolute refractory period it is not possible to generate another action potential. It occurs when most of the Na^+ channels are inactive during repolarisation. The epoch immediately after this is known as the relative refractory period. During the relative refractory period it is possible to generate an action potential but a larger than normal stimulus is required. This is because some of the Na^+ channels remain inactive and some of the K^+ channels are still open. Having generated an action potential at the axon hillock, it is conducted down the axon to the next synapse.

Synapse

A synapse is a chemical or electrical connection, found at junction between: two nerve cells, a nerve cell and a gland, or a nerve cell and a muscle fibre. The most common synapse in the human nervous system is chemical (Figure 7) and is characterised by the presence of a synaptic cleft, synaptic vesicles, and a delay due to neurotransmitter release. Chemical synapses are advantageous because they can be excitatory or inhibitory.

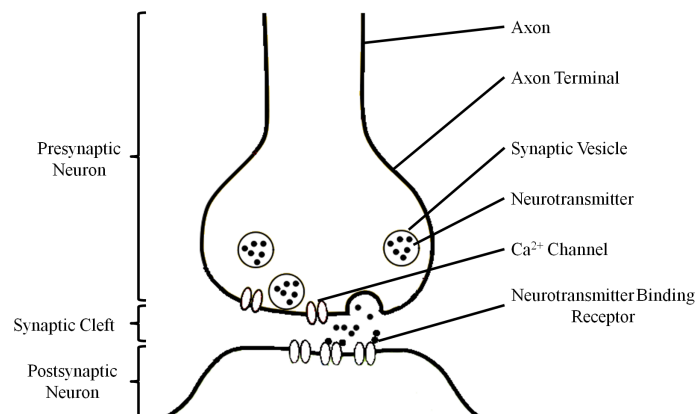


Figure 7: Schematic of chemical synapse (adapted from Branstrom *et al.* [183]).

Neuron-to-neuron synapses are typically found to be between: the axon terminal of the presynaptic neuron and the dendrite of the postsynaptic neuron (axodendritic synapse), the axon terminal of the presynaptic neuron and the soma (cell body) of the postsynaptic neuron (axosomatic synapse), or two axon terminals (axoaxonic synapse). Axodendritic and axosomatic synapses are similar because they directly carry input signals to the postsynaptic neuron. Axoaxonic synapses do not directly carry an input signal to the neuron, instead they can control the amount of neurotransmitter released by another neuron. Axoaxonic synapses can thus be excitatory or inhibitory.

An action potential arriving at the axon terminal opens voltage-sensitive Ca^{2+} channels, increasing the permeability of the terminal to Ca^{2+} , thereby permitting the flow of Ca^{2+} into the axon terminal. The influx of Ca^{2+} causes the synaptic vesicles to bind the membrane of the axon terminal and release a fixed amount of neurotransmitter into the synaptic cleft. The neurotransmitter diffuses through the synaptic cleft and binds to specific receptors on the postsynaptic cell membrane. This causes a change in the permeability of the cell membrane to particular ions, allowing them to flow, resulting in a polarisation of the membrane and a generation of a synaptic potential. If the synaptic potential is of sufficient magnitude, threshold will be exceeded and an action potential will be generated. Polarisation ends when the neurotransmitter dissociates from the the binding receptor. The neurotransmitter is then pumped back into the presynaptic axon terminal or is broken down by support cells and re-synthesised.

Synaptic Potential

Depending on the type of neurotransmitter released and the type of receptor binding site, the postsynaptic cell membrane will depolarise or hyperpolarise. Excitatory postsynaptic potentials are generated when the movement cations such as Na^+ and K^+ is allowed, resulting in an increased membrane potential and an enhanced postsynaptic potential. Inhibitory postsynaptic potentials are generated when the movement of anions such as Cl^{2-} is allowed, causing the the membrane potential to become more negative and resulting in an inhibited postsynaptic potential (Figure 8).

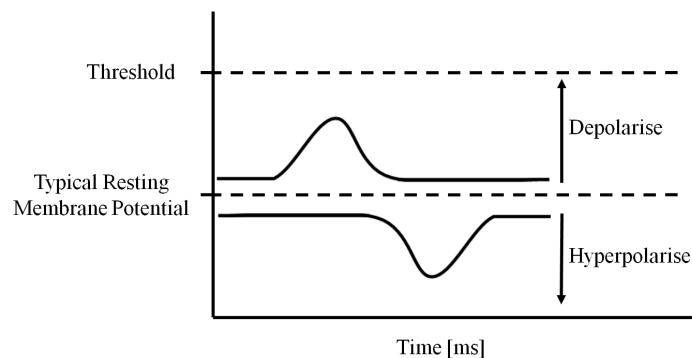


Figure 8: Schematic of a depolarising and hyperpolarising synaptic potential

Unlike the action potential, whose amplitude is always of the same magnitude, the synaptic potential is described as a graded potential. The magnitude of the synaptic potential is variable. It is dependent on the number of vesicles releasing neurotransmitter, which in turn

is related to the amount of Ca^{2+} entering the presynaptic axon terminal. Furthermore, a single synaptic potential is not of sufficient magnitude to reach threshold and generate an action potential at the axon hillock of the postsynaptic cell. For this reason the temporal, or spatial, summation of many incoming excitatory potentials is required.

Temporal summation occurs when several action potentials are delivered along the same axon but separated in time. The rapid firing of action potentials allows more Ca^{2+} to enter the axon terminal, releasing more neurotransmitter, which results in an enhanced post-synaptic potential. This is called potentiation. A similar effect is found with spatial summation, but in this case the action potentials are delivered from different axons rather than the one.

Inhibitory postsynaptic potentials can also be summed with excitatory postsynaptic potentials. Inhibitory signals counteract the excitatory action potentials and reduce the membrane potential to prevent threshold from being reached. The Renshaw cell is an example of an inhibitory neuron. It takes its innervation from the collateral of an α motoneuron and contains the neurotransmitter γ -aminobutyric acid (GABA). GABA permits the movement of Cl^{2-} across the postsynaptic cell membrane, thereby acting to reduce the overall membrane potential. Alternatively, the synaptic potential can also be reduced by presynaptic inhibition. This occurs when signalling at an axoaxonic synapse reduces the entry of Ca^{2+} to the presynaptic axon terminal.

Motor Unit Action Potential

The neuromuscular junction is the synapse between a nerve, such as the primary motor nerve (α -motoneuron), and a muscle fiber, and it operates in a similar manner to the neuron-to-neuron synapse (Figure 9).

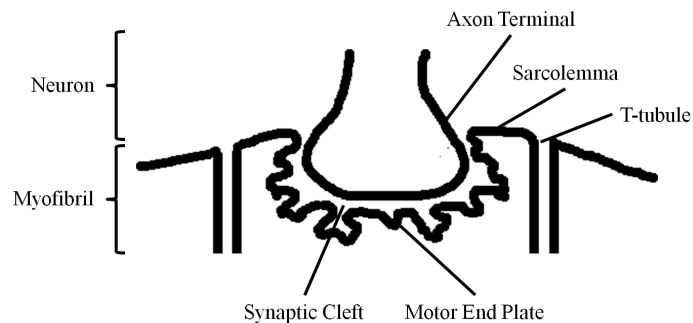


Figure 9: Schematic of a neuromuscular junction (adapted from Branstrom *et al.* [183]).

An action potential arriving at the axon terminal of the α -motoneuron opens voltage sensitive Ca^{2+} channels. The influx of Ca^{2+} into the axon terminal causes the synaptic vesicles to fuse with the axon terminal membrane. The neurotransmitter, acetylcholine, is liberated into the synaptic cleft and binds to specific receptors on the motor end plate. The motor end plate is a folded region of the sarcolemma, which itself is the membrane that surrounds the muscle cell. The binding of acetylcholine allows the influx of Na^{+} and efflux of K^{+} across the motor end plate, causing it to depolarise and generate an action potential. The action potential travels along the sarcolemma and down into the muscle cell via the t-tubules. Upon

entering the t-tubules, the action potential causes the terminal cisterna, which are reservoirs located on either side of the t-tubules to release Ca^{2+} ions into the surrounding intracellular fluid, called cytosol. The release of Ca^{2+} into cytosol generates a MUAP which propagates along the sarcolemma of the muscle cell causing it to contract, in a process described by the sliding filament theory. The MUAP ends when the acetylcholine dissociates from the binding receptor on the motor end plate.

A series of MUAP's occurring in relatively quick succession will result in a sustained contraction of the muscle fiber. The summation of several MUAP is described as a CMAP. Figure 10 below outlines how the summation of several MUAP in space and time result in a CMAP, which can be measured using EMG.



Figure 10: Example of the summation of several motor unit action potentials to give a compound motor action potential (adapted from Konrad [185]). The compound motor action potential here is representative of a typical electromyograph.

The motor nerve and muscle fibre that it supplies are one of the most basic functional units in the nervous system. Combined they form a motor unit. A contraction in skeletal muscle is detected by sensory receptors that relay information back to the nervous system regarding the position, location and orientation of the muscle in space, so called proprioception. The sensory receptors do this by measuring the length, change in length, and tension of the muscle.

Hardware Description

Electromyography: surface Electromyography (sEMG) was recorded using the 8-channel Bagnoli Desktop EMG system (Delsys Inc., Massachusetts, USA). An amplification of $\times 0$, 100, 1k, or 10k can be set on each channel. On-line cut-off frequencies are 20 ± 5 Hz (high-pass filter) and 450 ± 50 Hz (low-pass filter), and system noise within this bandwidth is $< 1.2 \mu V_{RMS}$. Single-differential, pre-amplified ($\times 10$ gain), surface electrodes (DE-2.1) with a parallel bar configuration and an inter-electrode distance of 10mm were used. A 50.8mm, circular, reference electrode (PALS Platinum, Axelgaard, Denmark) was used and placed in a region of low electrical activity, which was taken to be the lateral malleolus of the same limb. To ensure a high fidelity electromyograph, the skin of each recording site was prepared by gentle abrasion, using an abrasive gel (NuPrep, Weaver and Company, USA) and cleansing with an alcohol swab (UHS, UK). Recording electrodes were affixed to the skin using microporous hypo-allergenic surgical tape (3M Micropore, St.Paul, USA).

Accelerometry: Acceleration was recorded using tri-axial, $\pm 3g$, accelerometers (ADXL 335, Analog Devices Inc., Massachusetts, USA).

Goniometer: A linear potentiometer (Analog Devices Inc., Massachusetts, USA) was integrated into a knee brace and used to provide real-time visual feedback of knee angle, by displaying the voltage output on an oscilloscope.

Vibration: WBV was applied using a side-alternating vibration platform (Galileo Basic, Novotec Medical GmbH, Pforzheim, Germany). Vibration frequencies in the range of 12 - 27Hz, and vibration amplitudes in the range of ± 0 - 3.9mm can be applied. The amplitudes of vibration used in this study were guided by appropriate placement of the feet on designated markings on the vibration platform. Peak-to-peak vertical displacements at these markings are ± 1 , 2 and 3mm respectively.

Support: pBWS was provided using a body weight unweighing system (Loko S70, Woodway USA Inc, Waukesha, USA). An upper-body harness, fastened around the abdomen and strapped to the legs allows no- to full- body weight support.

Data Acquisition: All data was acquired at 1kHz, using a 16bit analog-to-digital converter (DAQCard-6036E, National Instruments Corporation, Texas, USA). Acquisition was synchronised using the Matlab Data Acquisition Toolbox, data was stored on a laptop pc, and signal processing was performed using the Matlab Signal Processing Toolbox, Curve Fitting Toolbox and Statistics Toolbox (MatlabR2010a, The Mathworks Inc., Massachusetts, USA).

Acceleration Profiles

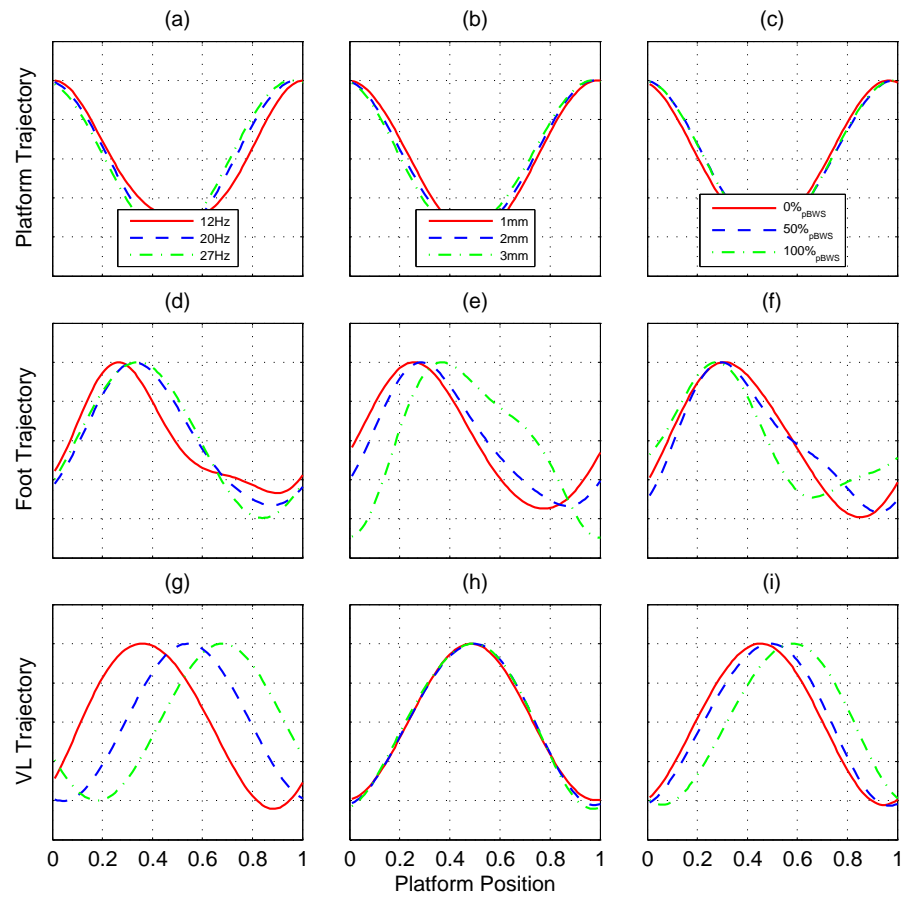


Figure 11: Platform trajectory along medial-lateral axis recorded from study with participants with no neurological deficit

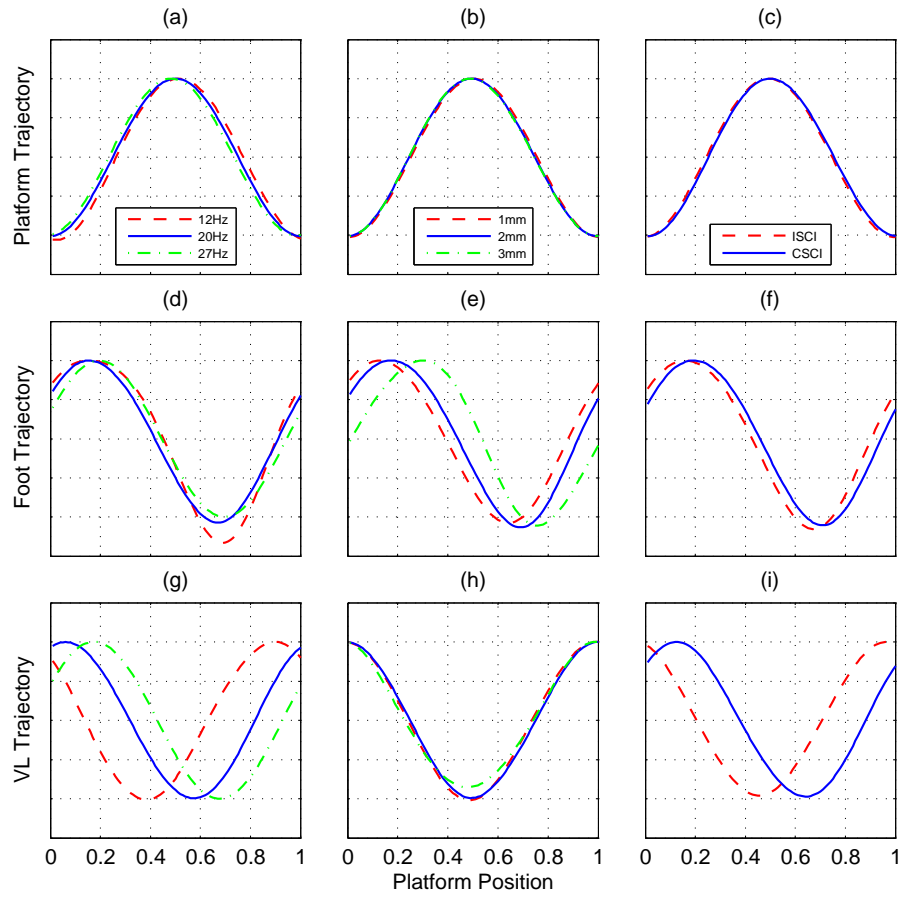


Figure 12: Platform trajectory along medial-lateral axis recorded from study with participants with a SCI

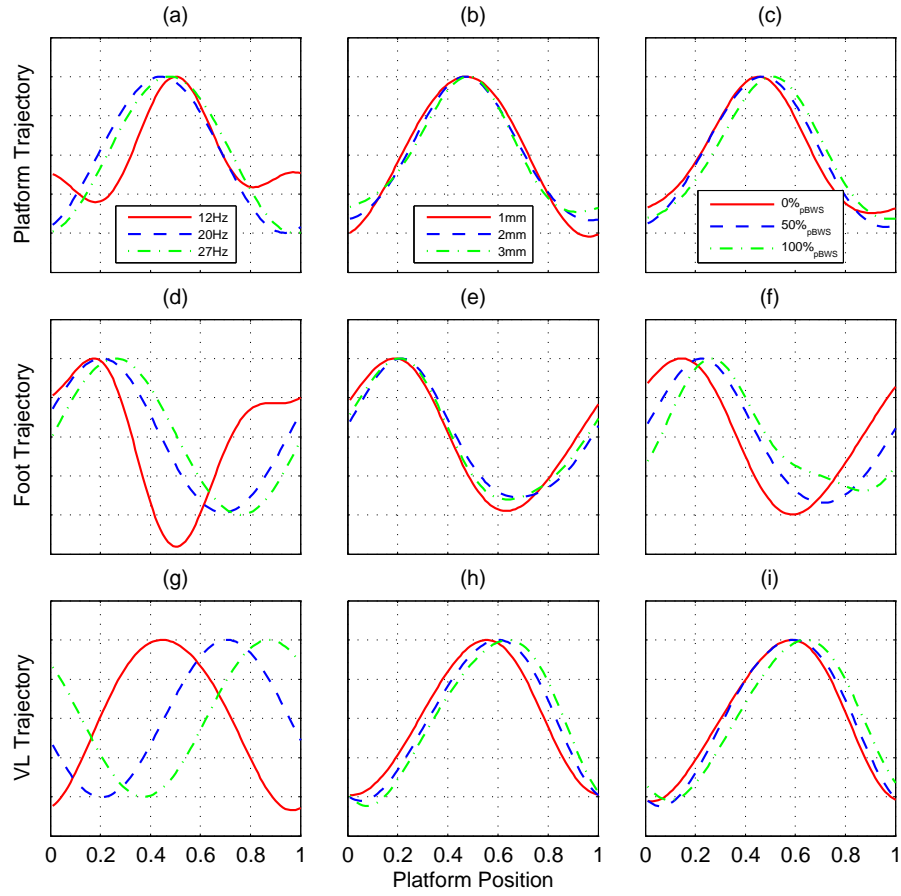


Figure 13: Platform trajectory along anterior-posterior axis recorded from study with participants with no neurological deficit

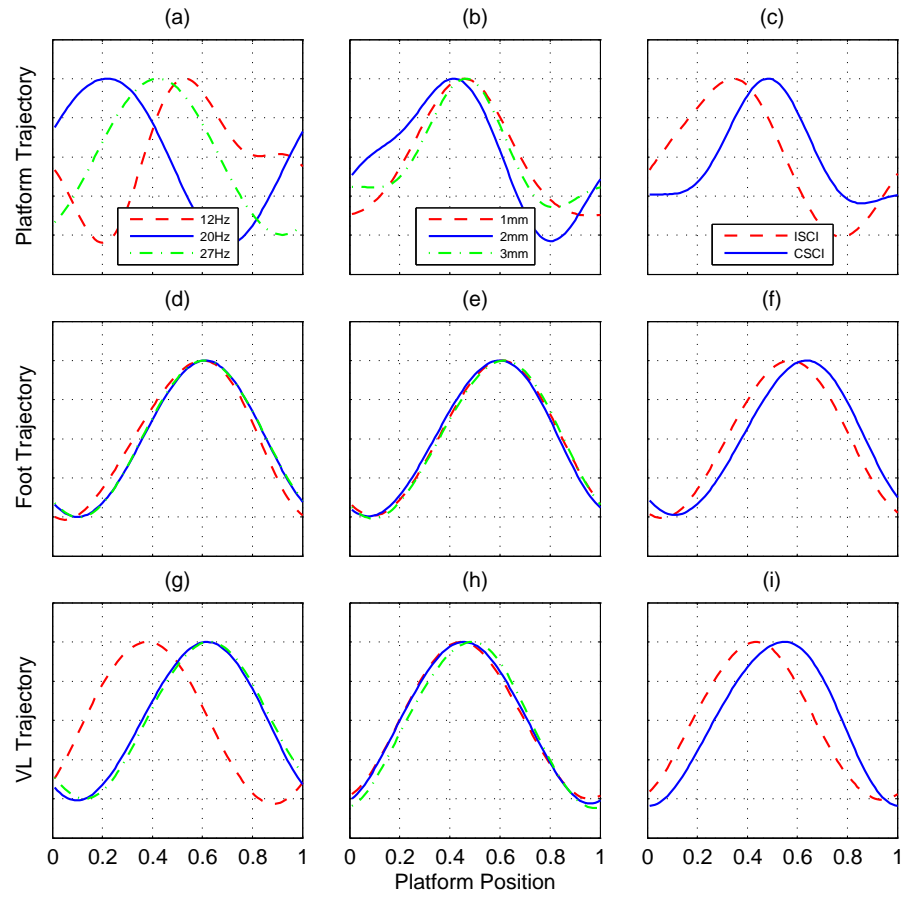


Figure 14: Platform trajectory in along anterior-posterior axis recorded from study with participants with a SCI

Research Ethics Approval Letters

WoSRES
West of Scotland Research Ethics Service

NHS
Greater Glasgow
West of Scotland REC 5
Ground Floor,
Tennent Institute,
Western Infirmary,
38 Church Street,
Glasgow G11 6NT
Tel: 0141-211-6270
Fax: 0141-211-1847

22 October 2009

Prof Kenneth J Hunt
Wylie Professor of Mechanical Engineering
James Watt South Building
University of Glasgow
Glasgow
G12 8QQ

Dear Prof Hunt

Study title: Cardiopulmonary responses during robotics-assisted stepping in acute spinal cord injury

REC reference: 08/S0710/66

Amendment number: Amendment 1.0

Amendment date: 21 September 2009

The above amendment was reviewed at the meeting of the Committee held on 21 October 2009.

Ethical opinion

The members of the Committee taking part in the review gave a favourable ethical opinion of the amendment on the basis described in the notice of amendment form and supporting documentation.

Approved documents

The documents reviewed and approved at the meeting were:

Document	Version	Date
Notice of Substantial Amendment (non-CTIMPs)	Amendment 1.0	21 September 2009
Covering Letter		21 September 2009

Membership of the Committee

The members of the Committee who took part in the review are listed on the attached sheet.

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Figure 15: Research ethics approval letter for study which investigated RATTT during early-stage SCI rehabilitation, page 1 of 3

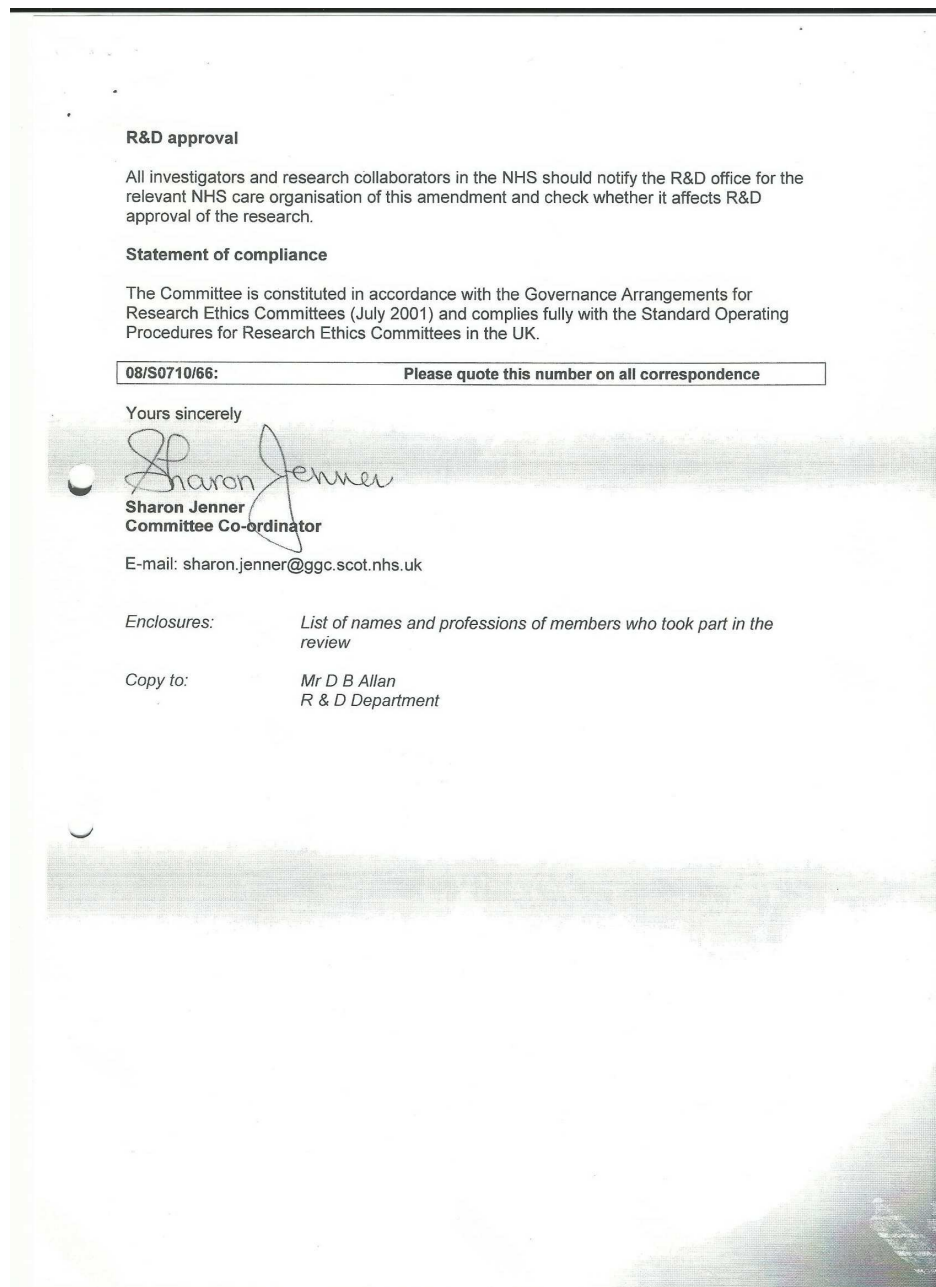


Figure 16: Research ethics approval letter for study which investigated RATT during early-stage SCI rehabilitation, page 2 of 3

West of Scotland REC 5

Attendance at Committee meeting on 21 October 2009

Committee Members:

Name	Profession	Present	Notes
Dr R Carelton	Consultant Psychiatrist	Yes	
Dr J Curran	GP	No	
Dr B Ellis	Head of Radiography	No	
Miss M MacCallum	Nurse Advisor	Yes	
Prof E McKenzie	Statistician	Yes	
Ms T McMichael	Health Promotion	No	
Mr A Morton	Lay member	Yes	
Dr G Ofill	Chair/Consultant Gynaecologist	No	
Dr A Rasul	Lay member	No	
Mrs J Russell	Lay member	Yes	
Dr W Smith	Renal Consultant	Yes	
Mrs L Tregonning	Vice Chair/Lay member	Yes	
Mrs E Griggs	Vice Chair/Lay member	Yes	
Mrs C R Hogg	Lay member	Yes	

Also in attendance:

Name	Position (or reason for attending)
Miss S Jenner	Co-Ordinator
Dr J Godden	Scientific Officer

Written comments received from:

Name	Position
Dr J Curran	GP

Figure 17: Research ethics approval letter for study which investigated RATTT during early-stage SCI rehabilitation, page 3 of 3

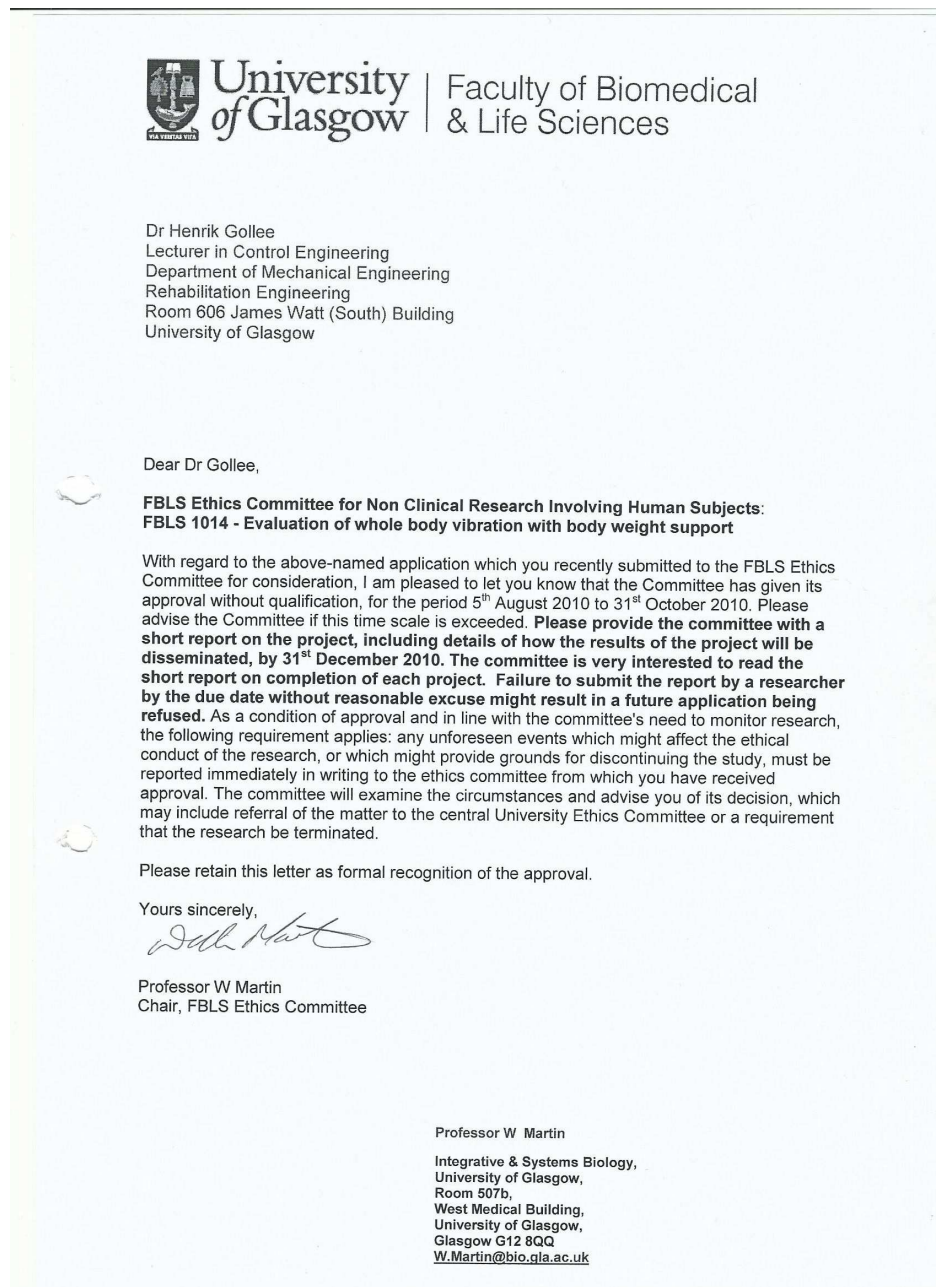


Figure 18: Research ethics approval letter for study which investigated WBV-pBWS in a general population, page 1 of 2

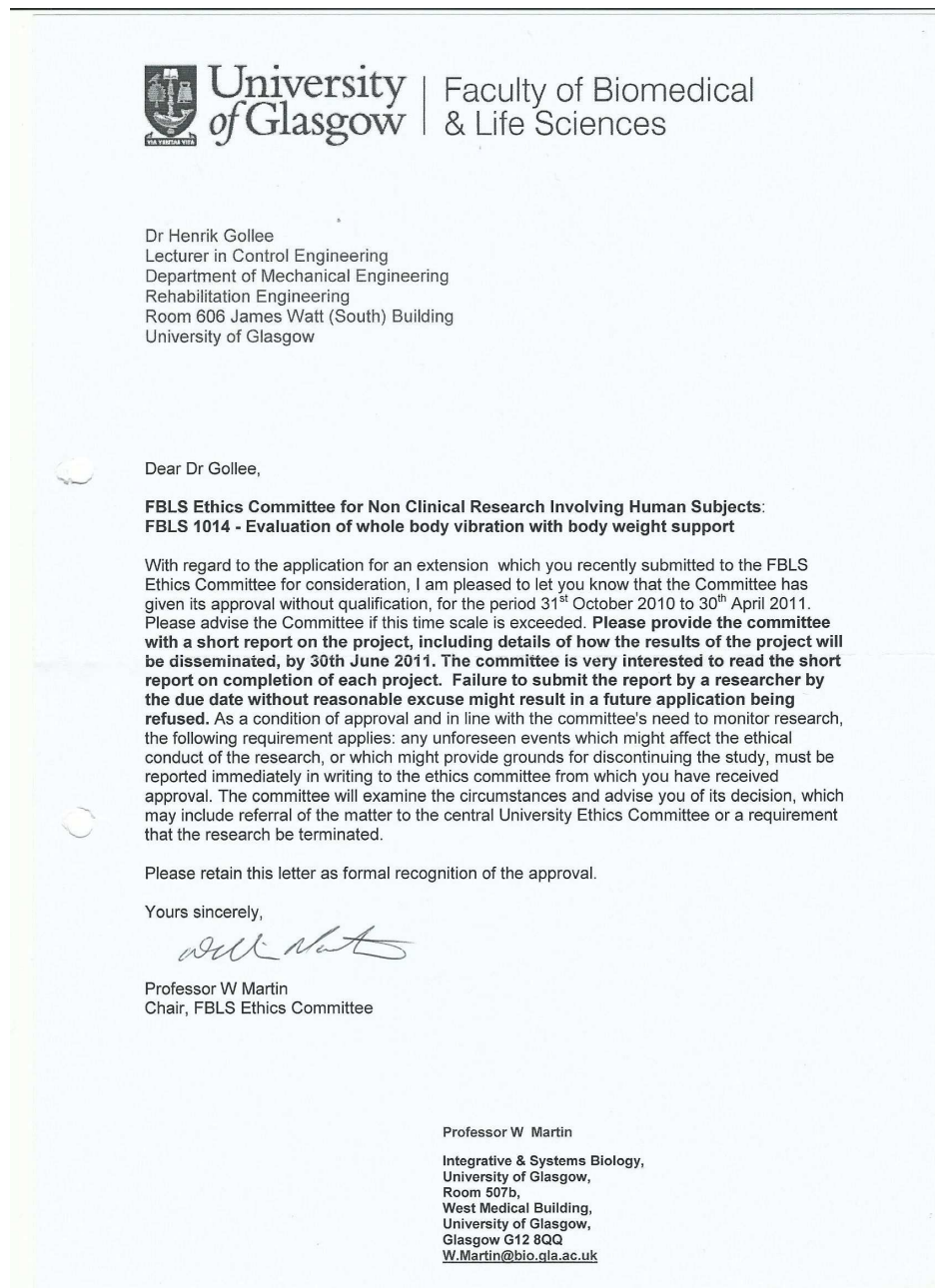


Figure 19: Research ethics approval letter for study which investigated WBV-pBWS in a general population, page 2 of 2



Figure 20: Research ethics approval letter for study which investigated WBV-pBWS in a SCI population, page 1 of 4

Management permission or approval must be obtained from each host organisation prior to the start of the study at the site concerned.

Management permission ("R&D approval") should be sought from all NHS organisations involved in the study in accordance with NHS research governance arrangements.

Guidance on applying for NHS permission for research is available in the Integrated Research Application System or at <http://www.rdforum.nhs.uk>.

For non-NHS sites, site management permission should be obtained in accordance with the procedures of the relevant host organisation.

Sponsors are not required to notify the Committee of approvals from host organisations

It is the responsibility of the sponsor to ensure that all the conditions are complied with before the start of the study or its initiation at a particular site (as applicable).

Approved documents

The final list of documents reviewed and approved by the Committee is as follows:

Document	Version	Date
Protocol	2.0	16 March 2011
Response to Request for Further Information		30 March 2011
Participant Information Sheet: Protocol 2	2.2	16 March 2011
Investigator CV		27 January 2011
Participant Consent Form	Version 1	20 January 2011
CV - Student - Mr Colm Craven		
Risk Assessment	1.0	30 March 2011
Participant Information Sheet: Protocol 1	1.2	16 March 2011
REC application		01 February 2011
Covering Letter		30 March 2011

Statement of compliance

The Committee is constituted in accordance with the Governance Arrangements for Research Ethics Committees (July 2001) and complies fully with the Standard Operating Procedures for Research Ethics Committees in the UK.

After ethical review

Now that you have completed the application process please visit the National Research Ethics Service website > After Review

You are invited to give your view of the service that you have received from the National Research Ethics Service and the application procedure. If you wish to make your views known please use the feedback form available on the website.

The attached document "After ethical review – guidance for researchers" gives detailed guidance on reporting requirements for studies with a favourable opinion, including:

Figure 21: Research ethics approval letter for study which investigated WBV-pBWS in a SCI population, page 2 of 4

- Notifying substantial amendments
- Adding new sites and investigators
- Progress and safety reports
- Notifying the end of the study

The NRES website also provides guidance on these topics, which is updated in the light of changes in reporting requirements or procedures.

We would also like to inform you that we consult regularly with stakeholders to improve our service. If you would like to join our Reference Group please email referencegroup@nres.npsa.nhs.uk.

11/AL/0096	Please quote this number on all correspondence
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With the Committee's best wishes for the success of this project

Yours sincerely

Liz Jamieson
Committee Co-ordinator
On behalf of Mr Eoin MacGillivray, Vice Chair

Enclosures:	List of names and professions of members who were present at the meeting and those who submitted written comments
	"After ethical review – guidance for researchers"
Copy to:	Mr Colm T D Craven Dr Erica Packard, R&D – NHS Greater Glasgow & Clyde

Figure 22: Research ethics approval letter for study which investigated WBV-pBWS in a SCI population, page 3 of 4

West of Scotland REC 3**Sub-Committee of the REC held in correspondence – deadline 07 April 2011****Members involved in the Review**

<i>Name</i>	<i>Profession</i>	<i>Present</i>	<i>Notes</i>
Mrs Bernadette Campbell	Primary Care Support Nurse	Yes	
Mr Eoin MacGillivray	Lay Member	Yes	
Dr Paul Mattison	Consultant Physician in Rehabilitation Medicine	Yes	
Dr Stephen Noble	Consultant Anaesthetist	Yes	

Figure 23: Research ethics approval letter for study which investigated WBV-pBWS in a SCI population, page 4 of 4

SCI Acceleration Analysis

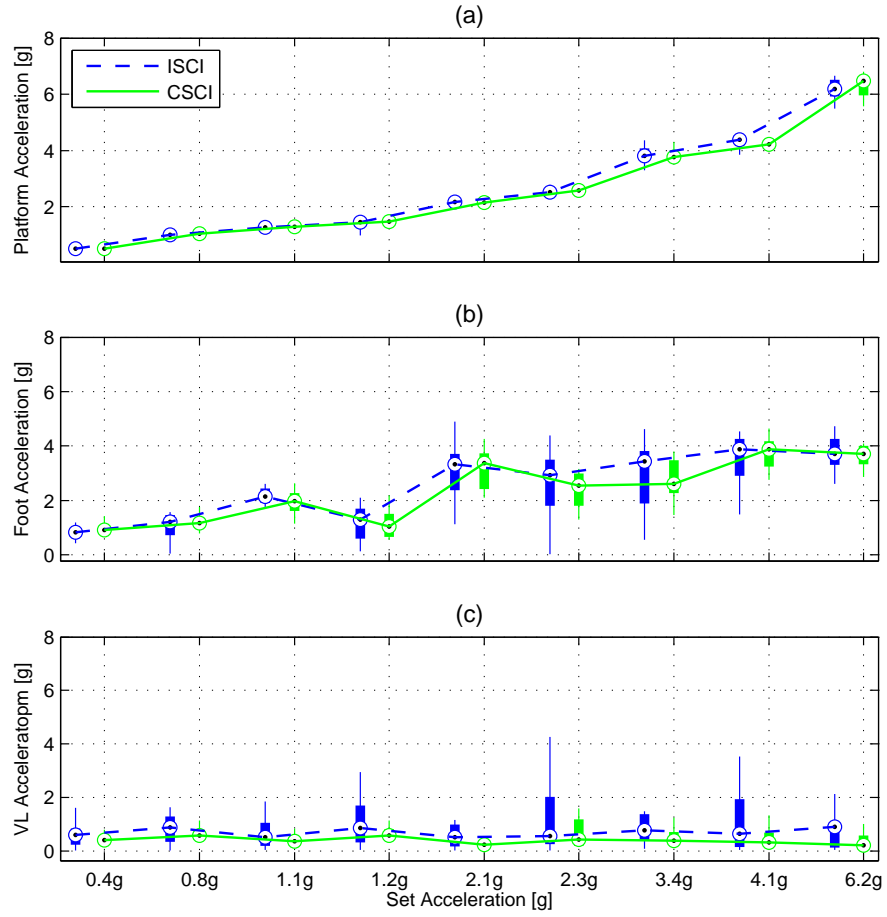
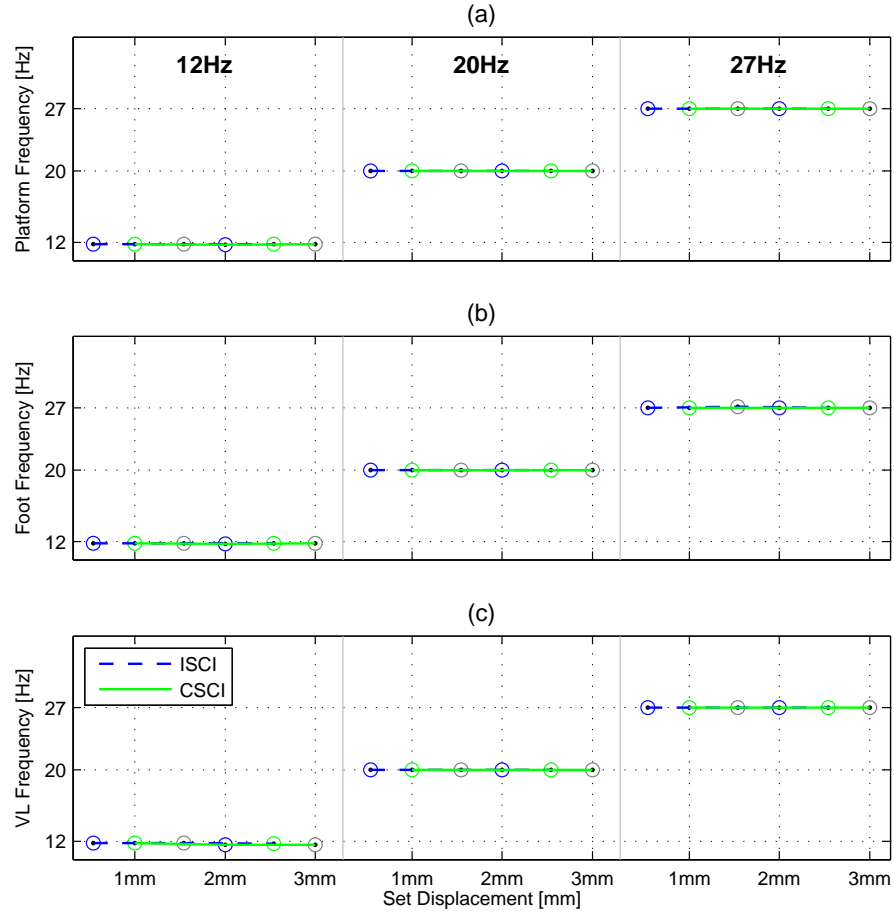


Figure 24: Average acceleration data recorded during WBV (Phase 2 from Figure 4.4 only) from the platform (a), foot (b) and VL (c). The data were grouped according to the expected platform acceleration (a_{theor}) which was calculated using Equation (4.13). The medians of the grouped data and the corresponding interquartile ranges are shown. Specific vibration frequency and vibration amplitude conditions may be identifiable by cross-referencing the a_{theor} values presented on the x-axis above with Table 4.6.



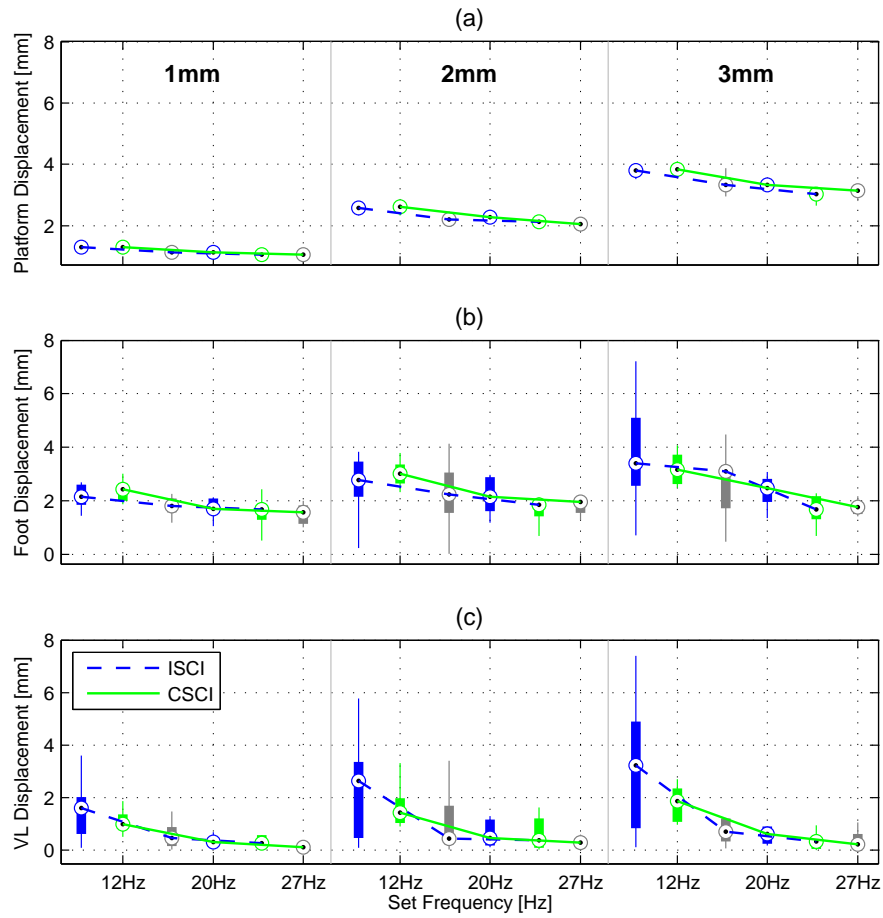


Figure 26: Displacement recorded during WBV (Phase 2 from Figure 4.4 only) from the platform (a), foot (b) and VL (c). The data were grouped according to vibration frequency and vibration amplitude. The medians and interquartile ranges of the grouped data are also shown.

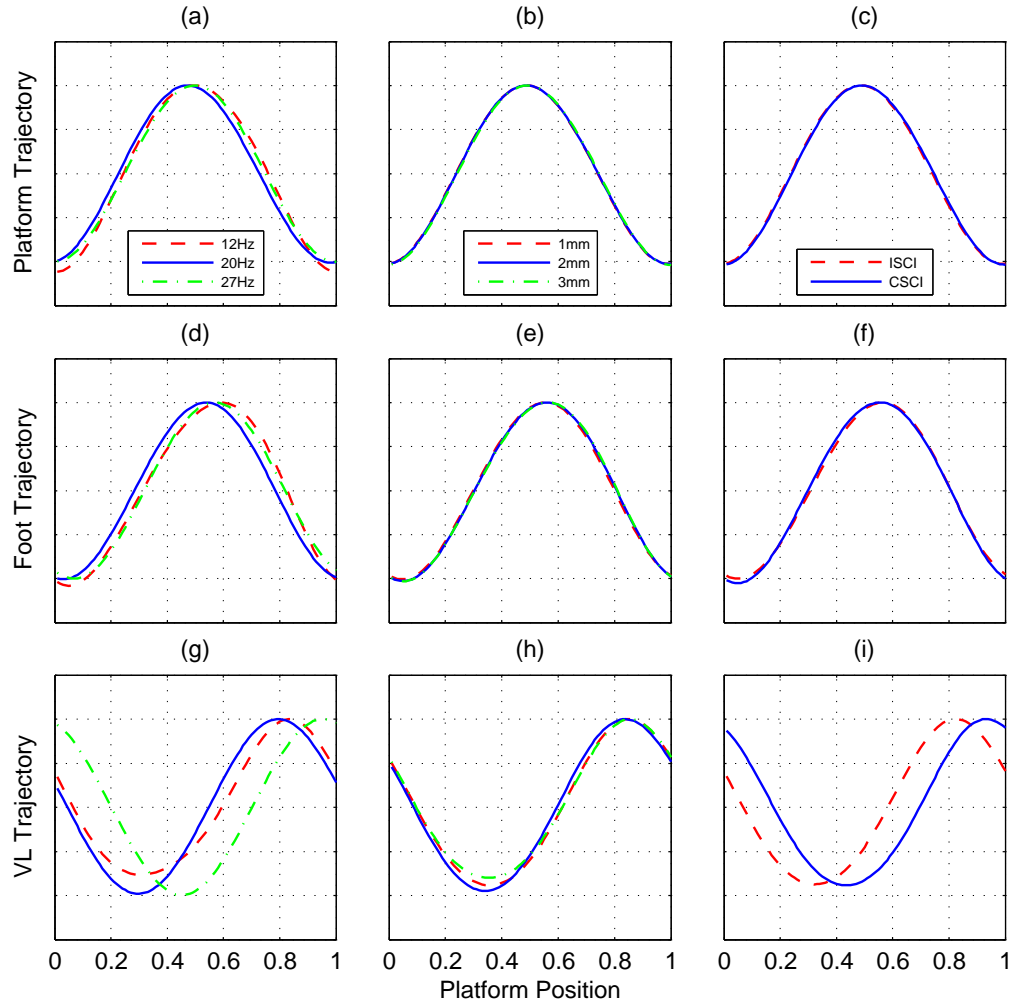


Figure 27: Platform, foot, and VL trajectories were estimated using longitudinal axis acceleration data recorded from each of these locations respectively during vibration. The data from each test was averaged for each cycle of vibration and normalised to the maximum acceleration value rendering them unitless. The data presented in (a), (d) and (g) were grouped according the vibration frequency and averaged for all participants, tests, displacements and classification of SCI. The data presented in (b), (e) and (h) were grouped according to vibration amplitude and averaged for all participants, tests, frequencies and classification of SCI. The data presented in (c), (f) and (i) were grouped according to classification of SCI and averaged for all participants, tests, frequencies and displacements.

SCI Artefact Analysis

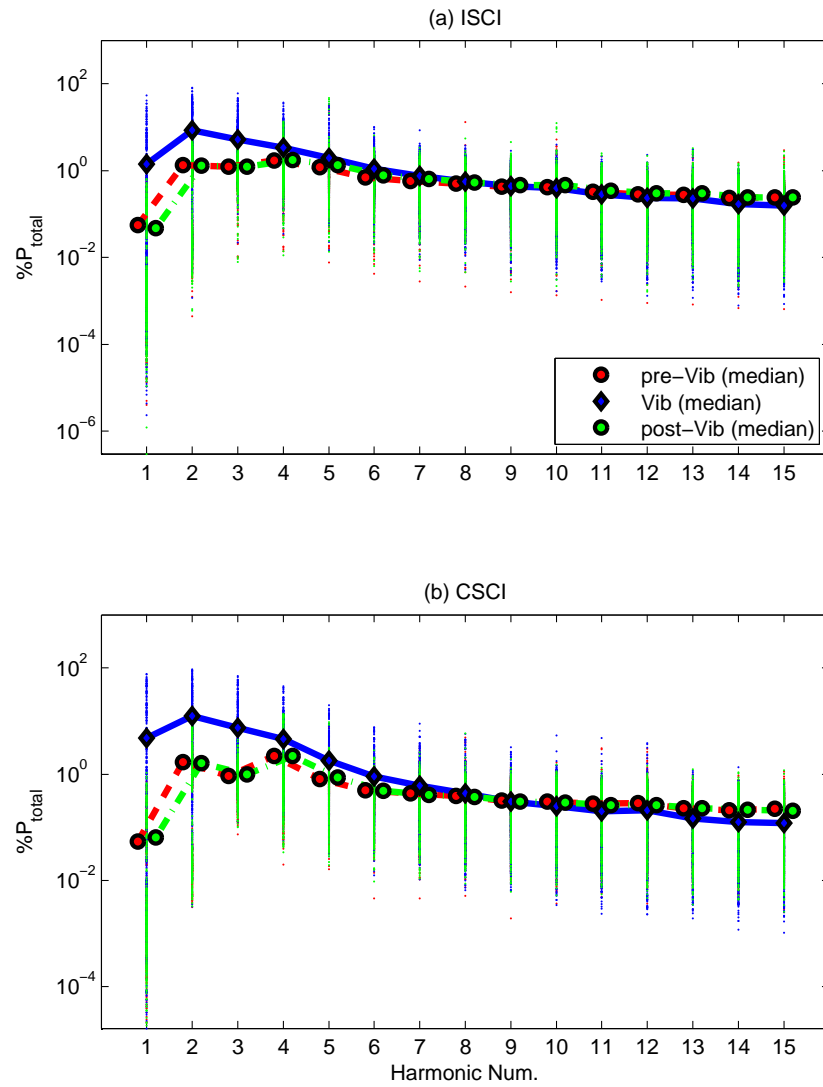


Figure 28: The power of the first 15 harmonics of vibration $\pm 1\text{Hz}$ are presented as a percentage of total EMG power (Equation (4.5)), for all SCI participants, sessions and test conditions. Each individual dot is a single data point. The data were grouped according to the harmonic number and the test epoch: pre-Vibration, Vibration and post-Vibration. The medians of each grouped data are also shown.

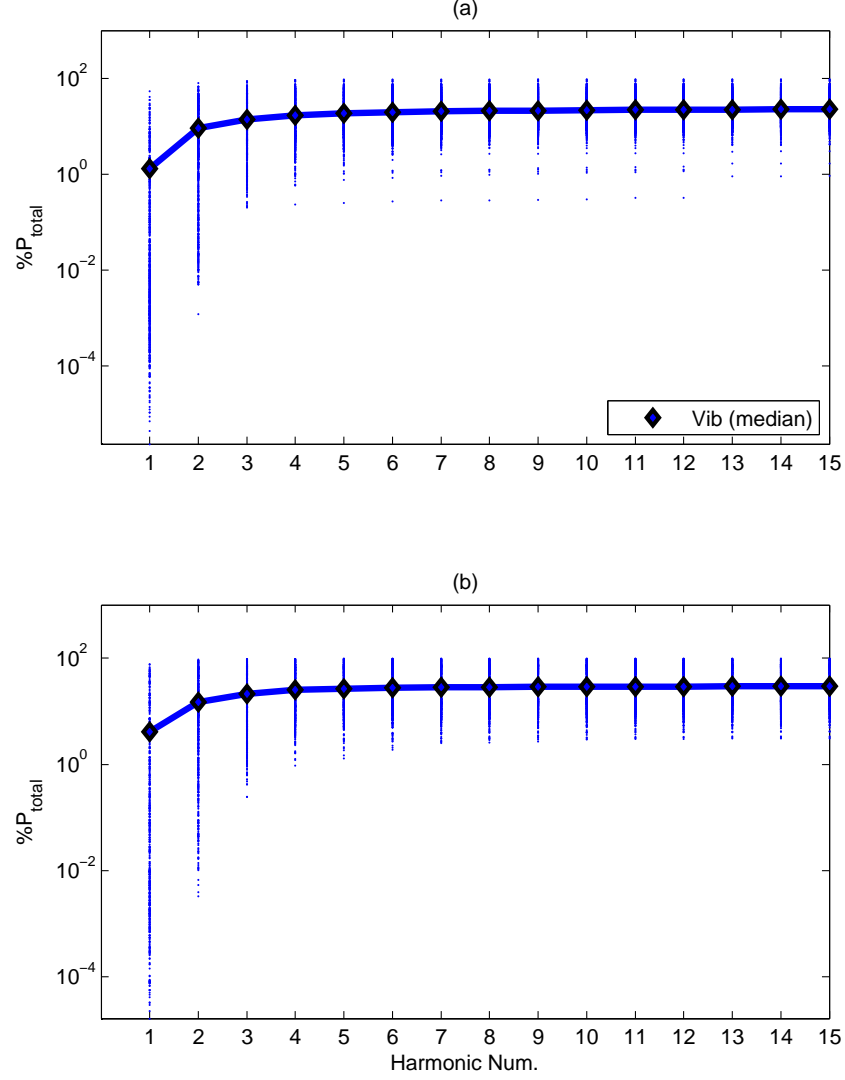


Figure 29: The integrated contribution of the individual harmonics p_{h_n} are presented as a percentage of total EMG power (Equation (4.6)), for all participants, sessions and test conditions. Each individual dot is a single data point. The data were grouped according to the harmonic number and the test epoch: pre-Vibration, Vibration and post-vibration. The medians of the grouped data are also shown.